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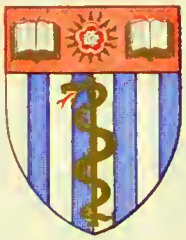
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NERVOUS DISEASES

AND THEIR

DIAGNOSIS:

A TREATISE UPON THE PHENOMENA PRODUCED BY DISEASES OF THE NERVOUS SYSTEM, WITH ESPECIAL REFERENCE TO THE RECOGNITION OF THEIR CAUSES.

BY

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TO

Surgeon JOHN S. BILLINGS, U.S.A.,

FACILE PRINCEPS OF MEDICAL BIBLIOGRAPHERS,

THIS BOOK

IS RESPECTFULLY DEDICATED, AS AN ACKNOWLEDGMENT THAT WITHOUT HIS SELF-SACRIFICING LABORS IT WOULD NOT HAVE BEEN POSSIBLE TO ITS AUTHOR, AND AS A TOKEN OF THE WARMEST PERSONAL ATTACHMENT AND ESTEEM.

PREFACE.

By way of apology for again trespassing upon the patience of the profession, the author of the present volume would say that the work is founded upon a hospital service continuous, except for one short period, for twenty-five years. Of this service fifteen years were spent in the medical wards of very large military or civil general hospitals, while for the last ten years nervous wards in the Philadelphia Hospital, aggregating one hundred and twenty-five beds, and the nervous clinic of the Hospital of the University of Pennsylvania, comprising over five hundred new cases annually, have been under the charge of the author. In his youthful days he also served as resident physician in an insane asylum, and more recently he has been connected with several such institutions as a consultant.

Notwithstanding this experience, the author would not have ventured to add a new book to the already long list of treatises upon nervous diseases had he not been strongly urged thereto by some of his former pupils, who insisted that the method of teaching which had been gradually evolved in the weekly clinics of the University Hospital was different from that commonly in vogue, and if followed out would give freshness to an old subject.

The defects of the work are perhaps better known to its author than they will be to any of its readers or critics, but they are not the result of lack of honest effort, and if the kindly judgment of brother practitioners should by any chance bring the work to another edition, whatever criticism it may receive

will be thankfully accepted, thoroughly weighed, and duly acted upon.

In conclusion, the author desires to acknowledge his indebtedness to Dr. George E. De Schweinitz for much assistance rendered in various ways and places, but especially in the anatomical portion of the book, in the chapter on Eye Symptoms, and in preparing the index. Thanks are also due Mr. Joseph McCreery for his extraordinarily intelligent and suggestive reading of the proofs.

University of Pennsylvania,
January 1, 1887.

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INTRODUCTION.

The primary meaning of the word "disease" is pain, uneasiness, or distress. In its derived or secondary significance it is defined by Webster as a deviation from health in function or in structure. In modern medicine the term has come to have a still more restricted technical meaning, being used to signify a simple or complex pathological process which is complete in itself. Thus, a simple enteritis is a disease; but the enteritis of typhoid fever is a part of a complex pathological process which constitutes the disease known as typhoid fever. When the term disease is, as it ought to be, used in this narrow sense, the diseases of the nervous system are far fewer than the number usually assigned by standard authorities upon the subject.

The symptoms produced by pathological processes or diseases depend upon their seat; and whilst it may be necessary to consider acute inflammation of the brain as a different disease from an acute inflammation of the spinal cord, the various parts of the nervous system are so interwoven that we cannot draw lines between the various affections. Thus, a chronic poliomyelitis affecting certain regions of the medulla oblongata gives rise to the so-called glosso-labial paralysis, but when affecting the lower portions of the spinal cord it produces progressive muscular atrophy. Not rarely, however, both regions are simultaneously attacked, when, in deference to an unscientific and misleading nomenclature, the patient is said to suffer from two distinct diseases.

Partly led by considerations such as those just stated, and partly as the result of the exigencies of clinical teaching, in the course of years I have gradually adopted a method of instructing students which has seemed to me more in accord with the every-day needs of the medical practitioner, and more apt to lead to a practical understanding of diseases of the nervous system, than is that adopted in the ordinary treatises upon the subject. When

a case offers itself for examination, the physician must needs travel from the symptoms back to the lesion, and not from the lesion to the symptoms. He does not say, this man has a clot in the brain, therefore he has hemiplegia, but he begins with the paralysis, and passes from it by the process of induction to the lesion. Hitherto the authors of text-books have travelled from the lesion to the symptoms. The present treatise is an attempt to follow the route which the practitioner must pass over daily.

Before entering upon the discussion of the individual symptoms produced by diseases of the nervous system it seems to me necessary to consider the subject of *neurasthenia*, or *nervous weakness*, because the symptoms of this state are so indefinite and fugitive that it is almost impossible to marshal them into order.

Much effort has of recent time been wasted in attempting to make of neurasthenia a disease. It is a bodily condition which is frequently associated with various chronic disorders, and not rarely coexists with perverted functional activity of the nervous centres, which perverted nerve-functions may, however, exist independently of any perceptible neurasthenia, and are not simply the outcomes of the neurasthenia. It is an habitual foundation for hysteria, chorea, insanity, and various nervous diseases, but may exist without the superaddition of any of them.

The onset of neurasthenia is always gradual, although at times the condition appears to develop with great suddenness. Under these circumstances, however, the explosion has been preceded by a long train of more or less overlooked phenomena: thus, a gentleman who had long suffered from the premonitory symptoms of neurasthenia was one day seized with violent vertigo, accompanied by such prostration of strength that he had to be taken home from the street in a carriage. The symptoms vary greatly according to the portion of the nervous system which is especially affected, and also to some extent according to the etiology of the attack. Nervous exhaustion may in the beginning affect the whole of the nervous system, or it may be at first purely local and coexist with general nervous strength. Many cases of spermatorrhœa are instances of the local form of neurasthenia, the sexual centres being primarily affected; but as in these cases, sooner or later, the whole of the nervous system becomes implicated, so in other forms of the disorder the exhaustion, at first local, finally, if

neglected, implicates the whole organism. There are not rarely cases of brain-exhaustion in which the symptoms are at first purely local. Almost always the cause of a local neurasthenia is excessive use of the part: thus, cerebral asthenia is usually the result of mental overwork, sexual asthenia of sexual excesses, etc. When to the intellectual fatigue are added the depressing effects of excessive anxiety or allied emotions, the symptoms from the first are more general. The exhaustion may affect chiefly a single function of the brain. As an instance may be cited the case of a postal clerk under my care, who has been accustomed to distribute five to eight thousand letters every day from a general mass into three hundred pigeon-holes, representing as many post-office districts, scattered over a large territory. As soon as the address is read there must be an instantaneous automatic recognition of the district to which the letter goes. It is at this place that, in the case now under consideration, the symptoms manifest themselves. Reading the address fails to produce immediate recognition of the locality to which the letter is to be assigned. Asked in what district such a post-office is, the clerk answers instantly, but seeing the address himself he hesitates, and sometimes balks so that he can distribute only about one-third as many letters as when in health. As in most cases of local nerve-exhaustion, in this patient some evidences of general implication exist, there being decided disturbance of the sexual organs.

A form of local neurasthenia which is frequently associated with brain-exhaustion is writer's cramp. I have repeatedly seen it come on as the herald of a general break-down; but under such circumstances the symptoms have usually not been those of typical writer's cramp: there have been not so much marked spasms as loss of power and distress in the arm on attempting to write.

In pure brain-exhaustion loss of the disposition to work is usually the first symptom, the sufferer finding that it constantly requires a more and more painful effort of the will to perform the allotted task. The basis of this difficulty is largely loss of the power of fixing the attention, and this by and by is accompanied by weakness of the memory. Disturbances of sleep are frequent. Various abnormal sensations in the head are complained

of. In most cases there is not absolute headache, but a feeling of weight or fulness, or an indescribable distress, usually aggravated by mental effort.

It is true that in some cases of very dangerous brain-tire cerebration is performed with extraordinary vigor and ease; the power of work is for the time markedly increased, and even the quality of the product may be raised; the patient may glory in a wild intellectual exaltation, a sense of mental power, with an almost uncontrollable brain-activity. It is probable, however, that these cases are not instances of pure neurasthenia, but that there is an active congestion of the cortical gray matter. It is certain that they are very prone to end in serious organic brain-trouble. In some cases of cerebral asthenia there are disturbances of the special senses, tinnitus aurium, flashes of light, and even the seeing of visions. Under these circumstances it is again probable that active congestion of the affected centres exists.

Severe cerebral neurasthenia may be associated with good spirits, but usually there is marked depression, and this perversion of function may finally go on to decided melancholy. The will-power, like all the other functional activities of the brain, is prone to be weakened; morbid fears may finally develop; and at last that which was at the beginning a simple brain-exhaustion may end in hypochondriasis or insanity. According to my own experience, such ending is, however, very rare, unless there is an inherited tendency to insanity.

Disturbances of sensation are common in neurasthenia, these disturbances taking the form in many instances of itchings or formications or similar minor ills. Neuralgia is often severe and its attacks frequent, but I am convinced that something more than simple nervous exhaustion is responsible for its production. I believe that there is a neuralgic diathesis or temperament which is often associated with neurasthenia, but may exist without it, and which probably has, at least in many cases, relations to a gouty ancestry. When such temperament exists, the neuralgic attacks are greatly aggravated by the coming on of neurasthenia. Hyperæsthesia and anæsthesia mark the line where simple neurasthenia passes into hysteria. The same also is true of the peculiar tenderness over the spinal processes of the vertebræ, which is especially frequent in women, and is the chief symptom

of the so-called spinal irritation or spinal anæmia,—an affection which I believe to be a form of neurasthenia allied to hysteria.

In neurasthenia disturbances of the sexual organs are very common; in women great pain on menstruation, ovarian irritation, the so-called irritable uterus of Hodge, are closely connected with general nerve-weakness. In not a small proportion of the cases of uterine disorders which are locally treated, I believe the local disease is largely the expression of the general condition. It is well known that masturbation and sexual excess in the male may produce an exhaustion of the nerve-centres especially implicated, and also a general nervous exhaustion. This is the common history of spermatorrhœa. It is no less true that a general neurasthenia may produce a local weakness of the sexual centres, with symptoms at least resembling those of spermatorrhœa,—namely, great irritability of the sexual organs, with a practical impotence due to immediate seminal discharge whenever coition is attempted. I have certainly seen this condition result from excessive intellectual labor when there has been no sexual excess, and at a time when the muscular strength was still good. Such cases may, perhaps, be distinguished by the fact that unprovoked emissions are not nearly so apt to occur as in true spermatorrhœa.

In cases of nervous exhaustion the efforts of the diagnostician are chiefly directed to determining the cause of the exhaustion. In a very considerable proportion of cases which have been sent to me as suffering from simple neurasthenia, chronic malaria, chronic diarrhœa, Bright's disease, or other serious organic affection has existed: it is therefore essential that in every case of alleged neurasthenia a thorough examination be made to detect latent chronic disease. When no such disease can be found, the cause of the neurasthenia should be recognized. In rare cases it develops in women, without perceptible cause, at pubescence or at the climacteric.

Overwork, especially overwork combined with worry, and even continuous emotional depression, unaided by excessive work, are capable of producing a pure neurasthenia. As Samuel Jackson was accustomed to say, in his lectures at the University of Pennsylvania thirty years ago, "Whenever the expenditure of nerve-force is greater than the daily income, physical bankruptcy sooner

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or later results." It is to be remembered that the nerve-capital of persons differs almost as widely as does their moneyed capital. There are numerous families many of whose members are neurasthenies from birth,—*i.e.*, are born with less power of creating nervous energy than is neeessary to meet the requirements of the ordinary duties of life. There is every grade of natural endowment between the most feeble person, seareely able to produee more nervous energy than is necessary for breathing, eating, and drinking, and the organism that is eapable of enduring ineessant toil. The development of neurasthenia is therefore not so mueh the result of a strain which is absolutely great, as of a strain whieh is excessive in its relations to the organism whieh has to bear it. I have seen not a few cases in whieh the neurasthenia has appeared to me to be an expression of premature old age. In sueh cases the rigid, atheromatous radial arteries occurring in a non-gouty or non-syphilitic subject have pointed to a similar exeessive ripeness of tissue throughout the body.

DIAGNOSTIC SYMPTOMATOLOGY

OF

NERVOUS DISEASES.

CHAPTER I.

PARALYSIS.

Paralysis.—The word paralysis may be correctly employed to signify loss of nerve-function, either motor or sensory. But in the present volume it will be used to mean loss of voluntary motor power, the term anæsthesia being employed to represent sensory palsy.

Paralysis may be either partial or complete: a partially paralyzed muscle is capable of contracting with less than its normal force in response to the will; a completely paralyzed muscle is incapable of voluntary movement.

Detection of Paralysis.—In a case of supposed paralysis it is necessary first to decide whether the alleged loss of power results from a true paralysis or is due to other cause. I have not rarely seen a patient suffering from an immovable joint diagnosed as paralytic, when the immobility was the result of the inflammation of the structures around the joint. Again, loss of power may be the result of pain arresting motion, as in a rheumatic muscle. Spasm may also cause loss of mobility, and sometimes a failure of execution supposed to be paralytic is due to loss of co-ordination. A true paralysis is to be distinguished by the loss of the power of moving, either partially or entirely, together with there being no pain on passive or active movement, and no sense of resistance experienced by the physician when moving the affected

part. A paralysis may of course be coincident with a local inflammation which produces pain and soreness, and still more frequently is it associated with spasm: under these circumstances careful examination during both passive and active movements may be necessary for the detection of the underlying palsy.

For the estimation of the degree of partial paralysis various instruments have been employed: of these the only one commonly used is the hand-dynamometer. The power of the legs can usually be judged of with sufficient accuracy by noting, the height to which the patient, when sitting in a chair, can raise the feet, the ability to get out of the chair, and the power of endurance during standing or walking. A foot-dynamometer may be useful for estimating small gains of power under treatment, but is rarely employed.

FUNCTIONAL PALSIES.

When paralysis has been found to exist, it is necessary to determine whether it is a true organic palsy, or whether it is assumed, hysterical, or reflex in its nature.

Reflex Palsies.—It is well known that paralysis in a distant part is in some cases closely connected with a violent nervous irritation, such as a wound in the nerve-trunk, stone in the kidneys, etc. To such cases the name of Reflex Palsy has been given. There are, however, two entirely distinct classes of cases which have been grouped together under this name. In most of the recorded cases the paralysis has developed gradually, and has undoubtedly been the result of a secondary organic disease of the nerves or spinal cord. Thus, a man receives a wound in the hand involving a nerve, and slowly, step by step, the arm loses its power and becomes livid and cold. Under such circumstances there is undoubtedly an ascending neuritis. Or from a stone in the kidney a paraplegia gradually develops, the result of a secondary myelitis. (See Subacute Paraplegia.) There are, however, cases, like those reported by Dr. S. Weir Mitchell (Injury of Nerves, Philadelphia, 1872), in which the wound of a nerve is followed at once by a distant palsy; or like those in which a paraplegia is at once removed by the removal of an irritation, such as an adherent prepuce. In these cases there can be no organic

disease of the nerve or cord, and it seems to me no more extraordinary that there should be a reflex palsy than that there should be a reflex spasm. To enter upon the theory of these affections is beyond the province of this book. A true reflex palsy can be recognized only by its history: so far as the paralysis itself is concerned, there are no positive diagnostic indications.

Assumed Paralysis.—A paralysis may be assumed. When this is suspected, the tone of the muscles should be carefully examined, because in most palsies not accompanied by spasm there is a peculiar relaxation of the affected part; and the absence of such relaxation would of course strengthen suspicion. Sometimes the deceit can be detected by watching the patient when off his guard. Etherization may be resorted to. If the palsy be assumed, the mask will usually be thrown off during the stage of semi-unconsciousness, and movements will be executed with the paralyzed limb. I have seen the nature of an assumed palsy made manifest by the patient's failing to counterfeit properly the peculiarities of the form of palsy which he was imitating. Thus, in an assumed hemiplegia the face was drawn towards the affected side.

Hysterical Palsies.—The detection of the nature of the hysterical palsy is sometimes a matter of great difficulty. In some cases the hysterical nature of the affection is revealed by paralysis of the bladder, the intestines, the rectum, or other parts connected with organic life. Of course in some forms of organic palsies such symptoms are naturally present; but their diagnostic importance consists in the fact that they are frequently seen in hysteria associated with hemiplegia and local palsies, which are rarely, when of an organic nature, accompanied by paralysis of the non-striated muscle-fibres of the visceral walls.

The presence of other distinct symptoms of hysteria, either in the past or in the present, is of importance. Nevertheless a violently hysterical person may be attacked by organic palsy, and I have also seen hysterical paraplegia occurring without other symptoms of hysteria, and without an hysterical history that could be made out. The hysterical palsy is apt to be transient and shifting in its character, to go and come suddenly, and not to conform in its minor phenomena with the sequences and coincidences of organic palsy: thus, the reflexes are excited when

they ought to be depressed, or remain natural when they should be affected; or sensory disturbances are present when they should be absent, or are situated in portions of the body not corresponding with the place where they ought to be in an organic palsy. An atypical case of paralysis should always be viewed with suspicion, especially when occurring in a woman. Moreover, it must not be forgotten that a man may suffer from hysteria.

An hysterical paralysis may so closely simulate an organic one in its phenomena and its surroundings that it will for a time be impossible to decide whether it is organic or hysterical. It is, therefore, necessary to discuss in detail the diagnosis of hysterical palsy during the study of each of the different forms of paralysis.

ORGANIC PALSIES.

All paralyses are best arranged for study under six heads:

First. General Palsy, in which the whole muscular system is involved.

Second. Hemiplegia, in which the palsy affects one side of the body, or at least the arm and the leg of one side.

Third. Paraplegia involving the legs.

Fourth. Monoplegia, in which one member of the body is affected in the greater part of its muscular structure.

Fifth. Multiple Palsies, in which two or more disconnected, distinct groups of muscles are paralyzed.

Sixth. Local Palsy, in which a single muscle or a single group of muscles tributary to a single nerve is affected.

GENERAL PALSIES.

A true organic general palsy is a rare condition, and of course, except under extraordinary circumstances, cannot be absolute or complete, because the patient necessarily dies so soon as the trunkal muscles which are connected with respiration are affected beyond a certain degree.

If, however, a lesion be so situated that it interrupts the passage of the nervous course between the cortical brain-centres in which the impulse of voluntary movement originates, but does not affect the pathway between the automatic respiratory centres

in the medulla oblongata and the respiratory muscles, a complete general paralysis of voluntary movement may be consistent with life: such condition I have seen in an animal when the medulla has been divided from the pons, but I have never witnessed it in man, in whom it must be infinitely rare.

Hysterical General Palsy.—Hysterical general palsy is exceedingly rare, but Bruecke has reported six cases in which the principal muscles of the back and those of the four extremities were paralyzed, and there are others in medical literature. The nature of such a general palsy ought to be recognized by its coming on suddenly without being accompanied by severe apoplectic or constitutional symptoms, such as mark a sudden attack of an organic general palsy, and by the presence of distinctly hysterical symptoms, or of a history of previous severe hysterical manifestations.

Alcoholic Palsy.—A general loss of motor power is one of the symptoms of chronic alcoholism; but, with the knowledge of the habits of the patients, a mistake can hardly be made as to the cause of the failure of muscle-power. In the great majority of cases the arms are affected before, or more profoundly than, the legs. The disturbances of sensation are very marked; indeed, except in rare instances, sensation is much more profoundly influenced in alcoholic poisoning than is motion, whilst the union of paralysis of sensation and of motion is excessively rare in organic general palsy: further, the peculiar tremors, the alterations of brain-function, also serve as sign-posts in alcoholic palsies.

There are cases in which it is difficult to diagnose between an alcoholic palsy and true general paralysis of the insane. Dr. Camuset reports (*Ann. Méd. Psychol.*, 1883, vol. x. p. 201) cases in which tremors, general loss of muscular power, delirium of grandeur, unequal pupils, fibrillary contractions about the mouth, and all the supposed characteristic symptoms of the organic disease, were present, but which resulted in cure upon the forced disuse of alcohol. The only test in these cases is the effect of abandoning alcoholic potations. (See General Paralysis of the Insane.)

Intermittent Palsy.—An intermittent general palsy may be produced by malarial poisoning. A case of this character has been reported by Cavaré (*Gaz. des Hôp.*, 1853), in which the paroxysms lasted from five to eight hours, were of the quotidian type, and

were cured by quinine. Romberg has reported a case of intermittent paraplegia also of the quotidian type and cured by quinine. There would appear, however, to be cases of intermittent general palsy not due to malarial poisoning. A most remarkable instance of this is that reported by Professor C. Westphal (*Berl. Klin. Wochenschrift*, 489), in which the patient suffered from a number of attacks, commencing with pain in all four extremities, with rapid loss of power deepening into almost complete paralysis, ending in twelve or fourteen hours in sleep, with complete return of power on awakening. But the most remarkable features in this case were, that the sensibility was normal; that the knee-jerk in the height of the paroxysm was wanting; and that the electrical reactions were greatly weakened, and in some of the muscles entirely disappeared. After the paroxysm the electrical reactions became normal, except that the contractions were weak in those muscles which had been most affected. This patient recovered in four weeks. A case similar in character to this was reported by Hartwig (*Inaug. Diss.*, Halle, 1874), but the electrical reactions of the muscle did not disappear, although they became weak. In this case quinine at first appeared to prevent the paroxysms, but finally failed of effect, although the patient had had tertian fever some years before. Seven and one-half months after the beginning of this disease the patient had not recovered.

Multiple Paralysis simulating General Palsy.—A widespread multiple paralysis may closely simulate a general palsy, and, indeed, in certain conditions of the body may be said to pass into a general palsy: if two-thirds of the muscular structure be involved in a multiple palsy, the symptoms closely simulate a general paralysis, and if more than this proportion of the muscles are attacked, the symptoms become those of general palsy.

Toxæmic General Paralysis.—An apparent general palsy which is the outcome of a multiple palsy is usually due to a toxæmia, either by alcohol or by such metals as lead or arsenic; but it may be produced by a multiple neuritis.

Ascending Paralysis.—The so-called ascending paralysis in its latter stages gives rise to a general palsy, the nature of which is to be recognized by the gradual but rapid spreading of the symptoms from the feet or the hands, or simultaneously from each towards the centre. The symptoms of ascending palsy in its

various forms will be fully discussed under the head of Subacute Paraplegias.

For diagnostic purposes Organic General Palsies may be divided into those which originate—

First. In the cerebral hemispheres.

Second. In the pons.

Third. In the spinal cord, including the medulla oblongata or the intracranial portion of the cord.

Cerebral General Palsy.—A general palsy of brain-origin is always incomplete, and is always associated with symptoms of profound alteration of other brain-functions. The lesion which produces it must affect both hemispheres of the brain, and is, when suddenly developed, almost invariably of the nature of a tremendous outpouring of blood into one hemisphere, or the rupture of an abscess, or a double apoplexy affecting each side of the brain. If the symptoms be slowly developed, they mark the presence of a very wide-spread and serious degeneration of the cerebral cortex, and, unless the attack dates back to infancy, are almost diagnostic of the so-called general paralysis of the insane. (See Disturbances of Intellection.)

In some rare cases *Spastic Infantile Paralysis* is so wide-spread as to take the form of a general palsy, although, properly speaking, it is a multiple palsy, under which heading its symptoms will be fully described. The character of such a pseudo-general palsy is to be recognized by the disease dating back to early childhood, and by the presence of distortions due to excessive muscular contractions with more or less atrophy of the affected parts. There is also more or less pronounced arrest of mental development.

Pons Palsy.—A suddenly-developed general palsy, due to lesion of the pons, is always the result of a hemorrhage which involves both sides of the pons. These cases are very rare, and are always accompanied by severe apoplectic symptoms. The recognition of a general palsy in severe apoplexy is excessively difficult, owing to the general muscular relaxation. Nothnagel affirms that there is no case in literature in which a patient has recovered consciousness with a double palsy due to hemorrhage in the pons.

A more or less pronounced general palsy may be gradually produced by a progressive lesion of the pons or by tumors pressing upon the pons. In a case recorded by Hallopeau (*Archives de Physiol. norm.*, 1876) the symptoms slowly involved first the right side of the body and afterwards the left, as the lesion progressed through the pons. In a similar case reported by Stein (*Memorabilien*, 1863, 198) disturbances of sensation preceded, and exceeded in prominence, those of motion.

The diagnosis of tumor of the pons must be made partially by exclusion: thus, an organic general palsy which is not of brain or of spinal origin is usually due to disease of the pons. The gradual implication of one side of the body after the other is very strong evidence that the lesion is in the pons. Disturbances of temperature, vomiting, a nearly complete facial palsy, or early implication of other nerves which arise in the pons, would confirm a diagnosis reached by exclusion. The characteristic symptoms may, however, be nearly or even altogether wanting.

Bulbar General Palsy.—Theoretically, a general palsy might be produced by a minute hemorrhage into the central portions of the medulla. The medulla is, however, so small and so filled with vital nerve-cells that hemorrhage into it usually produces death, either instantaneously or in a few moments,—a result which also follows thrombus of the basilar artery, which supplies the respiratory centre. I know of but one case in literature in which it has been proved by a subsequent autopsy that the patient had survived hemorrhage into the medulla. This case is reported by Hughlings-Jackson in the *London Lancet*, vol. ii., 1872, p. 770. Whenever a series of symptoms pointing towards apoplexy of the medulla oblongata is followed by recovery, the strong probability is that there has been a temporary arrest of circulation in the *anterior spinal artery* or in the *posterior* (inferior) *cerebellar artery*, branches of the vertebral artery which supply the medulla. Any arrest of the circulation in the branches which the *basilar artery* sends into the medulla is followed by immediate death, since these do not anastomose, but are terminal arteries supplying the respiratory centres.

People have been suddenly attacked with violent headache, giddiness, severe hiccough, various disturbances of sensation, great difficulty in or total loss of the power of swallowing and

speaking, consciousness being preserved and the attack being at once followed by a more or less pronounced general palsy, with various local facial paralyses and great disturbances of respiration. There has also, in these cases, usually been excessive dyspnœa and general cyanosis, ending in death. In such attacks the lesion is *thrombus* or *embolus* in the *vertebral artery*, with consequent softening in the medulla oblongata. In some cases of this character, when the anterior pyramids have escaped, there has been no paralysis of the limbs; and there have been instances in which only two extremities have been paralyzed. Anæsthesia has not been noted in any of the cases.

Inflammatory Bulbar Palsy.—An acute palsy which may involve one, two, or more of the extremities and give rise to a monoplegia, hemiplegia, or general palsy, may be the result of a very rapid localized myelitic process occurring in the medulla. It seems to me most probable that in these cases the alleged inflammatory change has been preceded by degeneration of the vessels, and consequent thrombus. However this may be, the diagnosis of an acute lesion of the medulla is to be made out, in these as in all similar cases, by noting the paralysis of one or more of those organs whose nerves arise in the region of the medulla. A full statement of these palsies will be given under the head of Local Paralyses.

A slowly-developed general palsy may result from a tumor or other progressive disease affecting the medulla oblongata, and Nothnagel asserts that it may be the only symptom of such a lesion. In such a case the absence of evidence of implication of the higher nerve-centres, and the presence of blindness or other symptoms of basal brain disease, might enable us to locate the lesion at the base of the brain, and yet it might at the same time be impossible to decide with certainty whether it was the pons or the medulla that was involved. Usually, however, the seat of the lesion is revealed by disturbance of the functions of the nerves which originate in or pass through the medulla: so that the absence of such disturbance indicates, but does not prove, that the pons is affected. Vomiting is, I think, more frequent in disease of the pons than in affections of the medulla.

Sensation in Bulbar Palsy.—Contrary to what might be expected, loss of sensation in the extremities is not usually promi-

nent in a progressive general palsy of bulbar origin, and I know of no case in which there has been pronounced anæsthesia. In some instances complaint has been made of a feeling of stiffness, formication, or numbness in the extremities. The lack of disturbance of sensation is probably due to the fact that usually the lesion is a tumor springing from the brain-membrane and pressing upon the medulla: moreover, if the lesion has its seat in the medulla, it is commonly situated in the anterior portion. In either case death must result before the lesion is sufficiently advanced to affect markedly the sensory region.

Vaso-motor disturbances have not been prominent in the reported cases of disease of the medulla, but diabetes insipidus and even true saccharine diabetes have been noted.

Lenticular or Corpus Striatum General Paralysis.—Sudden loss of power in the tongue, lips, and muscles of mastication and deglutition has occurred from hemorrhage into the lenticular nucleus of each hemisphere, and in other cases slower, bilateral changes in these nuclei have produced a more gradual development of these palsies, with a general loss of power, making a picture resembling that of bulbar palsy. To these cases has been given the name of Pseudo-bulbar Paralysis. In a case recorded in the *New York Medical Record* none of the extremities were paralyzed; but, as a lesion of the lenticular nucleus usually causes hemiplegia, it is clearly possible for a double lesion to cause a general palsy which may very closely resemble that caused by disease of the medulla.

HEMIPLEGIA.

A hemiplegia in its fullest development affects the arm, leg, face, and tongue; the paralysis in the arm and leg is complete; the paralysis of the face is incomplete, and usually affects almost solely the muscles of expression about the mouth, the upper portion of the face ordinarily responding to the will. The muscles of chewing are very rarely involved. The corner of the mouth is usually drawn towards the sound side, but the tongue protrudes towards the paralyzed side, owing to the intact genio-glossus muscle, which thrusts the tongue forward, not being opposed by its fellow. In old hemiplegics the face is sometimes drawn towards the paralyzed side by the late contractures in the paralyzed muscle, and

in acute hemiplegia, if the paralysis be accompanied by spasm, a similar distortion may occur. The palsy in these cases of facial distortion from secondary spasm is to be recognized by the general immobility of the paralyzed side, by the absence or softening of the natural wrinkles and lines of expression, and by the loss of the ability to close the eye. Sometimes, when spasm obscures the paralysis, on forced smiling the loss of power is evident.

The muscles of the trunk and of mastication are very rarely paralyzed in hemiplegia, unless the base of the brain or the medulla be involved. The ordinary respiratory movements continue, because the lesion is situated above the automatic respiratory centre. More than this, in most cases the muscles of the trunk respond to the will to a certain extent: frequently, however, when the patient contracts the muscles either of mastication or of respiration as powerfully as he can, it will be noted that the muscles upon the sound side act more vigorously than those on the paralyzed side. The most plausible theory which has yet been brought forward to account for the escape of the masticatory and trunkal muscles is that of Broadbent. The muscles which escape are always those that are habitually used together: thus, the two masseters in eating contract simultaneously, and the respiratory muscles of the two sides of the trunk always act in unison. It is believed that by this habitual action the pathways are opened between the centres in the spinal cord which control the muscles under discussion, so that these two centres finally act in unison, and when one of them receives an impulse from a hemisphere this impulse overflows to its fellow. For this reason, if the lesion occur in the left hemisphere the right hemisphere is able to reach by its impulse the centres on each side of the cord connected with mastication or with respiration.

According to Dr. W. R. Gowers, immediately after the hemiplegic stroke the sound leg sometimes distinctly shares in the weakness. This also is to be explained on the theory of habitual action. Dr. Gowers further states that in some cases of hemiplegia when the patient tries to show his teeth the mouth may be motionless on the paralyzed side, and yet on smiling there may be little or no difference in the action of the two sides. Dr. Gowers explains this by supposing that emotional movements are indifferently innervated from either hemisphere.

Hysterical Hemiplegia.—Hemiplegia is a very frequent form of hysterical palsy, but in most of these cases one extremity is distinctly more affected than the other, and paralysis of the face, although it does occur, is so rare that any case in which it is present is probably not hysterical. The palsy is rarely complete: so that a patient unable to walk or even to stand may be able to raise the foot when in bed. There is usually more or less pronounced loss of sensation in the paralyzed part, and the coexistence of a hemianæsthesia with hemiplegia should always incite to a very careful diagnostic examination. The faradic contractility is usually normal, but in some cases galvanic sensibility is lost. Such loss is a very strong indication that the attack is hysterical. The reflexes which are usually excited in the first days of organic palsy are in most cases of hysteria not affected, and may be lessened. By attention to the points just enumerated, and to the history of the patient, the diagnosis can usually be made without difficulty. In some cases aid may be obtained by the application of powerful electrical currents to the affected membranes, or by the practice of the so-called metallo-therapy. Rapid alterations of sensibility produced in this way are very characteristic of hysteria. Sudden changes in the degree of paralysis are almost pathognomonic of hysteria: they do occur in cases of brain-tumor, but not in cases of cerebral hemorrhage.

Diagnosis between True and False Hemiplegia.—True hemiplegia is usually the result of a focal brain-lesion, but it is necessary to distinguish carefully between a true hemiplegia and one in which a multiple paralysis affects various groups of muscles upon one side of the body. If a majority of the leg- and arm-muscles on one side are affected by a multiple palsy, the symptoms may very closely simulate those of hemiplegia, but the true nature of the affection can usually be recognized by the irregularities in the intensities of the palsies. In a brain-hemiplegia the law is that the nearer the muscles are to the trunk the less apt are they to be completely palsied. Thus, the forearm is more thoroughly affected than the upper arm, and the leg than the thigh. In multiple paralyses simulating hemiplegias this rule is almost invariably departed from. A hemiplegic multiple palsy may be due to disease of the brain-cortex, and might well be considered to be a double monoplegia,—*i.e.*, a brachial and a crural monoplegia.

In this case the rule concerning the relative condition of the muscles just laid down does not always hold, and it is not always possible to distinguish the nature of the affection except by the collateral symptoms, which in the multiple palsy will generally show either that the brain-cortex is affected, or that there are two distinct lesions. When a hemiplegia dates back to childhood, and is associated with marked contractures, it is usually a multiple brain-palsy. (See Spastic Infantile Paralysis, under heading of Multiple Palsy.)

Spinal Hemiplegia.—A hemiplegia may be of spinal origin, due to pressure of a tumor or to hemisection of the upper cord: such hemiplegia is rarely complete, and is almost always associated with hemianæsthesia, the paralysis of sensation invariably being *on the opposite side* of the body from that of motion. When cerebral hemiplegia and hemianæsthesia coexist, they are invariably on the *same side* of the body, except in very phenomenal cases in which the paralysis of motion and the loss of sensation are due to different foci of disease situated in opposite brain-hemispheres.

Cerebral Hemiplegia.—When in any case the existence of a true brain-hemiplegia has been determined, the next step is to find out whether it has developed suddenly or gradually.

Sudden or abrupt brain-hemiplegias may be due to cerebral hemorrhage, to arrest of cerebral circulation by an embolus or thrombus, to an abscess, or to a tumor. Progressive or slowly-developing hemiplegias are the result of abscess, localized cerebral softening, or other progressive focal brain-degeneration, including tumors and gummatous inflammation.

In a progressive hemiplegia very little can be learned from the palsy itself as to the nature of the lesion. A paralysis produced by softening is usually more steadily progressive and less variable than one caused by tumor; the hemiplegia of tumor is, in its turn, surpassed in its perpetual variability by a hemiplegia of specific origin.

Abrupt hemiplegias produced by tumors are usually the result of sudden local congestions around the tumor, and are to be distinguished by their incompleteness and by the rapidity with which they subside. Their diagnosis is especially to be made out, however, by the presence of headache, epileptic fits, choked disk, or

other evidences of progressive cerebral disease. When a complete and permanent hemiplegia occurs suddenly in a case of progressive brain-disease, it is usually due to hemorrhage. The absolute abrupt hemiplegia, which sometimes results from the bursting of an abscess in the ventricle, is apt to develop almost at once into a general palsy, and is always accompanied by stormy cerebral disturbance, such as profound coma, great alterations of temperature, convulsions, etc., and is followed by death in a few hours. If the previous history of the case be known, the nature of the attack can readily be surmised.

Abrupt Cerebral Hemiplegia.—An abrupt complete hemiplegia may be due either to a hemorrhage into the substance of the brain, to thrombosis, *i.e.*, arrest of the circulation by the formation of a coagulum inside of the artery, or to embolism, *i.e.*, arrest of circulation by the lodgment of a small mass swept from a diseased heart-valve or other focus into the general circulation.

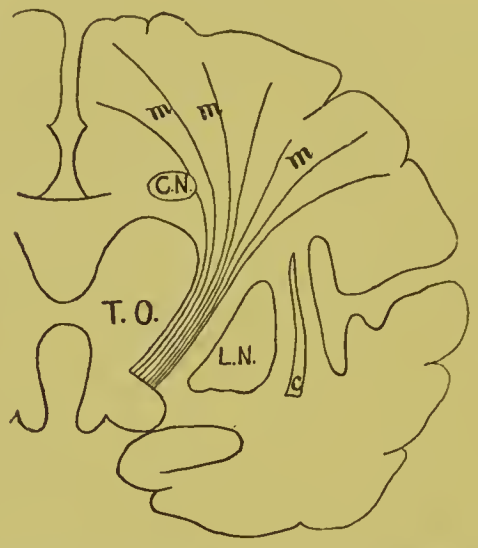
Diagnosis between Embolism and Hemorrhage.—In the great majority of cases the cause of a sudden hemiplegia is a rupture of a blood-vessel and the formation of a clot in the brain. A hemiplegia due either to a clot or to an arrest of circulation may occur with or without the symptoms of apoplexy. The diagnosis between hemorrhagic and embolic hemiplegia is often impossible. Various symptoms connected directly with the paralysis have been assigned from time to time as diagnostic of the arrest of cerebral circulation, but these symptoms are of no diagnostic value, and they shall not here be discussed. When the onset of a hemiplegia is accompanied by violent action of the heart, a full, strong pulse, and a general expression of great systemic power, it is almost invariably due to a clot; but a clot may be formed without such systemic reaction, and it is in such cases that the difficulty of diagnosis especially exists. It is asserted by some writers that hemiplegia accompanied by aphasia is usually the result of arrest of circulation; but in a number of autopsies upon such cases I have found a clot, and I believe that even this conjunction of symptoms is most frequently due to hemorrhage. When physical signs indicate the presence of numerous small vegetations upon the valves of the heart, or when these valves are undergoing rapid destruction, as in some cases of ulcerative endocarditis, a sudden hemiplegia may be very

reasonably suspected to be due to embolism. This suspicion is of course increased if previous embolic attacks, either of the brain or of other parts of the system, have occurred. Thus, in a case which came under my notice there had been previous attacks of violent local pains in various parts of the muscular system, associated with pronounced local disturbances of circulation, as shown by discoloration of the parts. It must also be remembered that large numbers of minute miliary aneurisms may exist in the brain and produce repeated attacks of slight hemiplegias, due to the formation of very slight clots outside of the vessels. If such a condition were coincident with cardiac disease it might very well be mistaken for embolic. According to Bourneville (*Études cliniques et thermométriques sur les Maladies du Système nerveux*, Paris, 1872), there is usually immediately after the formation of a thrombus a slight fall of temperature, followed by a slight rise, which may not pass beyond the norm, or in rare cases reaches to 40° C., and is apt to be accompanied by remarkable, irregular oscillations. Then there is usually a prolonged period in which the temperature remains nearly uniform, a little above normal and sometimes gradually rising, but not reaching a point equal to that very frequently attained in cerebral hemorrhage. These changes of temperature are not sufficiently different from those seen in cerebral hemorrhage to be of much diagnostic importance: they differ chiefly in intensity. The first fall and the subsequent rise of temperature are less than those which occur in hemorrhagic apoplexy, so that if in any case the thermometrical changes are very great, the symptoms are probably due to hemorrhage. A very great rise of temperature immediately after a hemiplegic apoplexy may be considered as pathognomonic of clot, and usually indicates a fatal termination. Bourneville has also noted that after death from acute brain-softening the temperature falls more rapidly than after cerebral hemorrhage.

Diagnosis of Position of Clot.— The diagnosis of the exact seat of a brain-lesion in hemiplegia involves a knowledge of the course of the motor fibres which run from the spinal cord to the cerebral cortex. The region of the cortex in which these fibres arise is so large that, unless by extraordinarily extensive lesions, only monoplegias or local palsies are produced. (See Local Palsies.) The motor fibres converge from the brain-cortex into a fasciculus

(see Fig. 1), which, in its entirety, is known as the *direct cerebral tract*, or as the *peduncular tract*. This band of fibres (*m*) runs between the external border of the thalamus optici (T. O.) and the lenticular nucleus (L. N.) of the corpus striatum, and constitutes a portion of the internal capsule so called. None of these motor fibres have been traced either to the optic thalamus or to the len-

Fig. 1.



Diagrammatic representation of course of direct cerebral tract. C. N., caudate nucleus; *m*, fibres of direct cerebral tract; *c*, claustrum; T. O., optic thalamus; L. N., lenticular nucleus.

ticular nucleus. The fasciculus passes from the brain-peduncles into the pons, and finally into the medulla oblongata.

Lesion of Thalamus Optici.—Theoretically, the only lesions in the interior of the brain which should produce hemiplegia are those situated in the direct cerebral tract. In accord with this, clinical records show that hemorrhages confined in their influence to the thalamus optici do not produce motor symptoms. It is true that often there is a temporary paralysis produced by hemorrhage into the thalamus, and that lesions of the middle third of the thalamus are not rarely accompanied by permanent hemiplegia. The middle third of the thalamus is that part of the ganglion in which the pressure from within would most affect the internal capsule and the pedunele. It would appear, therefore, that the paralysis in these cases is due to pressure upon the direct cerebral tract.

Lesions of Corpus Striatum.—Lesions of the corpus stri-

atum usually produce hemiplegia, which may be as complete as when the lesion is in the internal capsule, affecting the face, tongue, trunk, and extremities. Whether the paralysis in these cases is always due to pressure upon the internal capsule or not is an unsettled question. There is some reason for believing that the corpus striatum is in some way connected directly with voluntary motion. At present there is no method of diagnosis between lesions in the internal capsule and those in either portion of the corpus striatum,—*i.e.*, in the caudate nucleus or the lenticular bodies.

Facial Palsy.—In all cases of hemiplegia in which the lesion is in the central brain the paralysis of the face is upon the same side as that of the body.

Disorder of Sensation in Hemiplegia.—A lesion in the central brain very rarely affects sensation: for a discussion of such cases see the chapter on Disturbances of Sensation. In the medulla oblongata, the pons Varolii, the peduncles, and also in certain portions of the internal capsule, the motor and the sensory fibres are sufficiently close to be involved in a common lesion. In the peduncles slowly-growing tumors are the only lesions that produce purely motor hemiplegia: under such circumstances it is not possible during life to locate the lesion with certainty.

When hemianæsthesia and hemiplegia are produced by a focal brain-affection they coexist upon the side of the body opposite to the lesion, since the injury is situated above the decussation of both the motor and the sensory fibres. The detailed discussion of the exact position of the lesion in such cases must be deferred to the chapter on Disturbances of Sensation.

Hemiplegia with Aphasia.—A clot in the brain may give rise to hemiplegia with aphasia: the diagnosis of the seat of the lesion in these cases will be fully discussed in the chapter on Aphasia.

Hemiplegia from Lesion in Pons.—A hemorrhage into the pons Varolii may produce a simple hemiplegia which cannot be distinguished from one in the central brain-region; often, however, there are distinctive symptoms. In the small space occupied by the pons, there are gathered together motor and sensory fibres, as well as fibres belonging to the facial, hypoglossus, abducens, and trigeminus nerves: the symptoms of a clot in this region

may be, therefore, very various. It is, however, exceptional for an acute lesion of the pons to affect other than the facial nerve and the general motor tract. When the abducens nerve is implicated, there is an internal squint; it depends upon the portion of the pons attacked whether the misdirected eye is upon the side of the lesion or opposed to it: thus, if a right-sided lesion be in the upper half of the pons the squint will be in the left eye, but if in the lower half, in the right eye: in like manner in hypoglossus paralysis the tongue when projected turns from or towards the paralyzed extremities according as the lower or the upper portion of the pons is affected.* According to Nothnagel, a conjugated palsy of the abducens and the internal rectus muscle is diagnostic of lesion of the pons. General oculo-motor palsy can occur only when the lesion is a tumor sufficiently large to press upon parts near to but outside of the pons.

Facial Palsy.—In the majority of cases of hemorrhage into the pons, facial palsy is either wanting altogether, or is upon the same side of the body as are the other paralytic symptoms. Under these circumstances it is the proximal half of the pons—*i.e.*, that which is next to the peduncle of the cerebrum—that is affected.

Small hemorrhages or other lesions in the lower portion of the pons may cause hemiplegia without paralysis of the facial nerves; if the hemorrhage be at all large, there will be paralysis of the facial nerve opposite to the affected arm and leg. This alternate or crossed paralysis is produced by a lesion in the lower half of the pons,—*i.e.*, the half nearest the medulla oblongata,—because a lesion so situated is below the decussation of the facial nerves, but above the decussation of the general motor tract. The facial palsy produced by apoplexy of the pons is usually more complete than that caused by lesions in the cerebral hemisphere, but even in it the frontal and orbicular muscles almost invariably escape. In crossed paralysis the fibres of the facial nerve are affected below their origin in the facial nucleus. This nucleus belongs really to the spinal system, and is a trophic as well as a motor centre. Consequently, in alternate palsy the facial muscles are separated from

* The effect of palsies of these and all other individual nerves will be discussed in detail in the section on Local Palsy.

their trophic centres, and undergo degenerations similar to those which occur in the muscles of the extremities when the spinal trophic centres are involved. The nature of these degenerations will be discussed in detail in the section on Trophic Changes. It is enough for the present to state that, as first observed by Prof. Rosenthal (*Wiener Med.*, Halle, 1863), the facial nerve rapidly loses its faradic excitability, and develops an excessive sensibility to galvanic stimulation.*

Crossed paralysis is usually due to a lesion in the lower half of the pons, but is not absolutely diagnostic of such lesion, as it may be caused by a lesion in the medulla oblongata. Thus, Rondot reports (*Journ. de Méd. de Bordeaux*, vol. xiii. 304) a case in which softening was confined to the left pyramidal tract in the medulla, and H. Senator (*Arch. für Psych. u. Nervenk.*, xi. 3) one with softening extending from the calamus to the restiform body, due to thrombus of the left vertebral artery.

Crossed Oculo-Motor Palsy.—There are certain cases of hemiplegia in which the face, arms, and leg are paralyzed upon one side, although the oculo-motor nerve is affected on the opposite side of the body, as is shown by dilatation of the pupil and ptosis. In these cases there are usually temporary or permanent disturbances of sensation on the side of the hemiplegia. Very frequently there are marked disturbances of temperature, the paralyzed side being from two to nearly five degrees warmer than the normal side. The lesion under these circumstances is in the cerebral peduncle. (For cases, see Ramey, *Revue de Méd.*, 1885, 402.) Although Budge and Afasanieff place the centre which presides over the contractility of the bladder in the peduncles, it is rare for the rectum or the bladder to be affected in peduncular hemorrhage. It is much more frequent for œdema or reddish coloration of the skin to show evidences of vaso-motor disturbance.

Anæsthesia in Lesion of Pons. — Anæsthesia, usually absent, may be present in lesion of the pons. So far as the extremities are concerned, it always affects those which are paralyzed. In some cases hyperæsthesia of the paralyzed part has been noted directly

* Although this change of electrical relation has been noticed by several observers, I know of no recorded case in which the exact seat of the lesion has been confirmed by an autopsy.

after a hemorrhage into the pons, but it always disappears in the course of two or three days.

Professor Leyden has described a case of left hemiplegia in which a high grade of anæsthesia existed in the course of the right trigeminus nerve, and also in the left extremities; and Hughlings-Jackson states that he has seen a similar crossed sensory palsy. It would appear, therefore, that there may be alternate sensory as well as alternate motor palsy in disease of the pons. Under these circumstances it is probably the lower half of the pons that is affected.

Progressive Hemiplegia from Lesion of Pons.—A progressive hemiplegia with or without sensory disturbances, and with or without paralysis of the abducens, hypoglossus, or trigeminus nerves, may be produced by a slowly progressive lesion on one side of the pons. The diagnosis of such an affection must be made out by an application of the facts and principles which have just been discussed in detail in the consideration of acute pons lesions. As already stated, the gradual conversion of a hemiplegia into a general palsy is very characteristic of a tumor in the pons or in the membranes beneath it. I know of no studies of temperature under these circumstances; but very marked differences in temperature of the two axillæ, and especially in the temperature of exposed extremities, are to be looked for. In any case of progressive hemiplegia with a persistent marked increase of the temperature of one axilla, the probabilities are that the lesion is on one side of the pons.

PARAPLEGIA.

Paraplegia is a more or less complete palsy confined to the lower limbs, and may be either functional—*i.e.*, reflex or hysterical—or organic, it being understood that for the present we are forced to class under functional paraplegia cases in which after death no lesion can be demonstrated by the microscope.

Functional Paraplegia.

Reflex Paralyses.—Paralysis of a single group of muscles, or more usually of a number of associated groups, may result from the irritation of peripheral nerve-filaments not immediately con-

nected with such muscles. Instances of this are the various atrophic palsies associated with traumatisms, inflamed joints, and other surgical affections, which will be discussed in detail under the heading of Multiple Palsy. Omitting these surgical cases, the most usual form of reflex paralysis is paraplegia. For many years it has been known that paraplegia is not rarely associated with severe organic diseases of the genito-urinary organs, and in 1864 Brown-Séquard showed that similar loss of power in the legs may be produced by irritations of the intestines or other viscera, and gave to the affection the name of Reflex Paralysis. It does not lie within the scope of the present work to enter upon a detailed discussion of the pathology of renal paraplegia. Suffice it to state that in my opinion the more serious cases are the result of an organic disease of the cord (see page 44). This explanation, however, cannot be given of the not extremely infrequent cases in which a complete paraplegia without anæsthesia has occurred in a person suffering from a genito-urinary irritation and has disappeared within two or three days after the removal of such irritation. To cases of this character the name of Reflex Paraplegia should be restricted. The point of irritation may be in the intestines, and it is possible that in some of the cases in which paraplegia occurs during a severe dysentery the symptoms are reflex.* Usually, however, the paraplegia persists long after the

* Almost any form of paralysis may be developed during the convalescence from the acute fevers or exanthemata proper. There may be nothing in the paralysis to distinguish it from cases of similar character produced by other causes. Experience has, however, shown that in a considerable proportion of these post-febrile or post-exanthematous diseases the symptoms are simply a motor paraplegia, and are much more amenable to treatment than in ordinary paralysis of the same class. I have seen various instances of paraplegia following acute dysentery and typhoid fever in which I was unable to detect symptoms different from those produced by ordinary very mild myelitis located in the motor tract, but in which more or less perfect recovery took place in the course of a few months. The lesion in some of these cases is a multiple neuritis (see Multiple Neuritis); but that a myelitis may occur, and even be fatal, after one of these fevers, is shown by the case reported by Westphal (*Arch. für Psychiat.*, Bd. iv., 1873–74.) In this case the paralysis appeared on the eleventh day of smallpox, and resulted fatally in four weeks. At the post-mortem small foci of softening were found throughout the cord. It is probable that the myelitis was septic. Cases of dysenteric paraplegia have also been reported in which the autopsy has revealed diffused myelitis.

cure of the dysentery or diarrhœa, and the disease must therefore be looked upon as something more than a reflex affection. My belief is that in some of these cases there is organic change in the cord, but that in others there is only a condition of profound depression of the spinal function from loss of nutritive tone. The facts that in most cases the symptoms gradually yield to treatment, and that sensation is rarely if ever seriously affected, indicate that there is no serious myelitis. A true reflex paraplegia may be produced by the irritation of worms in the intestinal tract. A number of cases have been reported in which the passage of lumbricoid or tape worms has been followed by immediate relief of the paralytic symptoms. Dr. Moll, of Vienna, has reported a case in which the arms, and not the legs, were paralyzed, with an immediate cure of the palsy on the expulsion of the tape-worm.

In reflex paraplegia sensation is not disturbed, the bladder is not paralyzed, and there are no trophic changes.

Major Renal Paraplegia.—There have been not a few cases of genito-urinary disease, and especially of renal calculus, in which symptoms far more severe than those just spoken of were present. The motor paralysis in such cases increases until it becomes almost complete, and is accompanied with marked perturbation of sensation. Not rarely violent pains shoot down the affected limbs, and are associated with various paræsthesiæ, and a continually deepening anæsthesia which may become complete. In the earlier stages the reflexes are sometimes exaggerated, but sooner or later they grow less active, and in most cases finally disappear. The muscles rapidly waste, and the electrical reactions of degeneration appear. Bullæ, bed-sores, and other trophic changes increase: all control over the bladder and rectum is lost, and the patient finally dies from exhaustion. In some cases the progress of the disease is very rapid; in others it is slow, and arrest with partial recovery may occur if the original irritative lesion be removed. The symptoms in these cases are due to a secondary myelitis, which in some instances is certainly produced by a neuritis creeping up the nerve-trunk implicated in the original disease, and finally reaching the cord itself. I am inclined to believe, however, that a myelitis may be induced without this ascending neuritis, or in a manner parallel to that in which the condition of violent functional excitement of the spinal cord known as

tetanus is caused by an irritation of a peripheral nerve-filament. This secondary myelitis may be produced by any sufficiently severe and permanent irritation of nerve-filaments. Cases have been reported in which it has followed a direct traumatism of a nerve-trunk.

In some cases of pelvic or abdominal inflammations in which paraplegia has been supposed to be reflex, the symptoms have been due to a direct implication of the sacral nerve in the lesion, and a consequent wide-spread neuritis of the lower extremities.

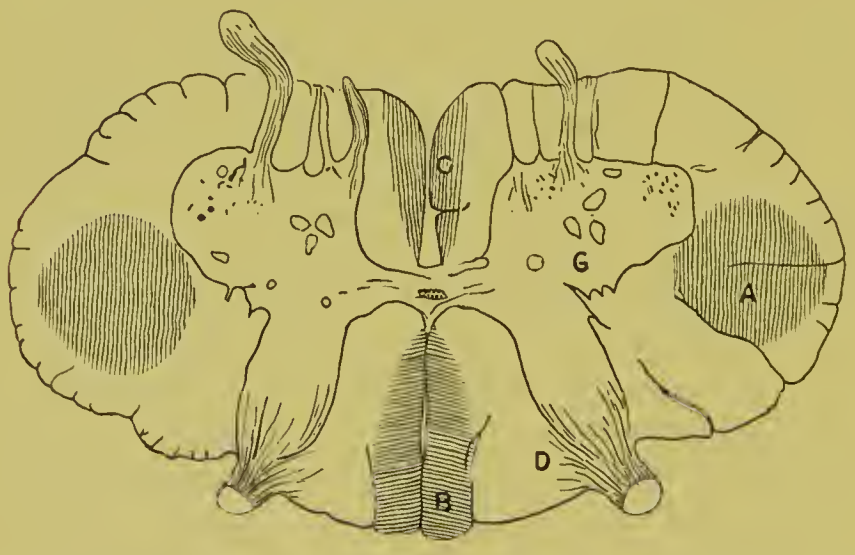
Hysterical Paraplegia.—Hysterical paraplegia is frequent, and may simulate any of the organic varieties. It usually develops rapidly, but may come on slowly. It may be associated with the most marked muscular relaxation, or with the greatest rigidity, due to excessive contractures. The knee-jerk (see Reflexes) is in some cases normal, in others it is absent. Perhaps in the majority of instances it is exaggerated. The muscles do not undergo rapid trophic changes, but a slow progressive wasting of them may occur. It has been asserted by Gowers that the existence of ankle clonus (see Reflexes) is proof of the organic nature of a paraplegia; but this is not correct. I have seen a paraplegia which had lasted for many months, associated with greatly exaggerated knee-jerk and pronounced ankle clonus, get well in a few days during the administration of subnitrate of bismuth. The most characteristic symptoms are connected with sensibility: in some cases there is excessive hyperæsthesia, with or without pain; more frequently the sensibility is lessened or abolished; usually the muscular sense is at least as much affected as is cutaneous sensibility. In those forms of organic paraplegia which are most frequently simulated by the hysterical affection, sensibility is not altered. According to my own experience, a distinct girdle sensation is diagnostic of organic disease; but hysterical patients are very prone to take on suggested symptoms: consequently they frequently complain of the girdle sensation after it has been mentioned in their presence. M. Charcot appears to believe that the presence of fibrillary muscular contractions is diagnostic of organic diseases, but this is denied. (See *Revue de Méd.*, 1885, p. 229.) The diagnosis of hysterical paraplegia is usually to be made out by considering the past history of the patient, the mode of onset, the condition of the sensibility, and

the shifting nature of the alterations of motility and of sensibility. It is also asserted, but on this point I am not positive, that in those cases of hysteria in which there are contractures,—cases in which the difficulty of the diagnosis is usually greatest,—muscular relaxation takes place in the early stages of etherization, whilst in the organic spastic palsy the anæsthetic exerts little or no influence on the muscles. Unless the diagnosis can be made by the use of an anæsthetic, there are certainly cases in which it is impossible to decide, within a brief space of time, whether the paralysis is organic or hysterical.

Organic Paraplegia.

Anatomy of the Spinal Cord.—In order to recognize the different forms of organic paraplegia it is necessary to have a clear understanding of the physiological regions of the spinal cord, and of the functions connected with each of these regions. In the following diagram it will be noticed in the first place that the

Fig. 2.



cord is composed of gray and white matter, and that in the gray matter of the cord are situated certain cells whose places are marked in the diagram by dots.

These cells are furnished with long processes, which are the origin of nerve-roots. They are ganglionic in their nature, and have the double power of exciting motion and of influencing nutrition in the muscles. When a disease attacks these gan-

glionic cells, paralysis of the muscles ensues, with rapid wasting and change in the electrical reactions. (See Trophic Changes.)

Placed laterally to the gray matter are the so-called *lateral columns* (A, Fig. 2), masses of nerve-fibres, which pass along the cord, constantly receiving accessions from the nerve-roots, and in the upper part of the medulla oblongata become the *pyramidal tracts*, which, crossing over to the opposite side, pass through the pons Varolii into the peduncle and then upward as the *direct cerebral tracts*. Situated on the extreme borders of the anterior fissure in the white matter of the cord are the so-called *columns of Turck*,—composed of white nerve-fibres passing upward to the brain (C). Their functions are similar to those of a lateral column,—namely, to conduct impulses from the brain. The essential difference is that the lateral columns cross over to the opposite brain-hemisphere, whilst the columns of Turck pass directly to the hemisphere of the same side: hence the lateral columns are sometimes spoken of as the *crossed pyramidal tracts*, and the columns of Turck as the *direct pyramidal tracts*.

From the gray matter of the cord pass out the anterior and posterior nerve-roots. The region of white matter in the neighborhood of the posterior roots is known as the *posterior root-zones*, and is connected with sensation and co-ordination, so that in disease of this portion of the cord these functions are especially affected. In immediate contiguity with the posterior fissure are the small tracts of white matter known as the *columns of Goll* (B). A disease which attacks the posterior root-zone usually affects also the columns of Goll, but there are very few cases on record of primary diseases of the columns of Goll: so that their functions are at present not definitely known.

Out of the spinal cord spring nerve-fibres, which pass into the so-called sympathetic ganglia. Such fibres arising in the cervical spinal cord pass through the cervical sympathetic ganglia, and go with the carotid artery into the cranial cavity. Some of these fibres are probably distributed as vaso-motor nerves of the brain and its membranes; others reach the eye, and become connected with the movements of the pupil. Certain spinal sympathetic filaments pass from the cervical dorsal region to the heart, and are essentially connected with its movements. It is owing to these facts that diseases of the cervical dorsal cord are

so frequently associated with derangements of the pupillary and cardiac movements.

In the lumbar cord are placed centres which preside over the genito-urinary tract, and hence disease of this portion of the cord is prone to be connected with priapism, impotence, or other genital symptoms, and with very early spasmodic or paralytic affections of the bladder.

Paraplegia from Multiple Paralysis.—When a multiple palsy attacks the lower portion of the spinal cord especially, it may produce a paraplegia which might be mistaken for one due to general myelitis. Under these circumstances, however, it will usually be found that some muscles of the lower extremities have escaped, or that they have been irregularly affected. In a myelitic paraplegia the general rule is that the muscles farthest from the trunk are first paralyzed; although this does not apply to cases of transverse myelitis or to some rare instances in which localized regions of the cord are especially attacked. Almost always in these cases of multiple palsy some muscles in the upper extremity will be found to be affected. The diagnosis of the true disease, however, is to be especially based upon the rapid wasting of the affected muscles and the change in their electrical reactions. Cases of multiple paralysis which most resemble myelitic paraplegia are those due to lead or arsenical poisoning. That such cases belong in the multiple palsies is shown by the trophic changes which are present and by the irregularities in the grouping of the palsies. The diagnosis between these cases and those of ordinary poliomyelitis or of multiple neuritis will be fully considered under the head of Multiple Palsies.

Organic paraplegia having been found in any case, the first point to be settled is as to the length of time required for the development of the symptoms. For diagnostic purposes all these cases are arranged under three heads:

First. Those in which the symptoms are developed with great rapidity.

Second. Those in which some days are required for the full production of the paraplegia.

Third. Those in which the symptoms progressively increase during a period of many months or years.

Abrupt Paraplegia.

When the symptoms of paraplegia develop in the course of two or three days, and are not connected with a traumatism, they are due either to a hemorrhage into the cord, to a hemorrhage into the vertebral canal outside of the cord, to ascending (or Landry's) paralysis, or to a very acute myelitis.

Spinal Apoplexy.—In some cases of sudden paraplegia the patient falls to the ground. Very rarely the cerebral disturbance is so marked that the attack may appear to be a true apoplexy; but when consciousness is restored it will be found that there is a complete palsy of the lower limbs, both of sensation and of motion. In the very beginning of the attack there may be violent pains, but these soon subside. Theoretically, spinal apoplexy might be expected to produce localized palsies in the parts below the lesion, but practically the cord is so small that whenever hemorrhage does occur it influences the whole of the cord, so that both sides of the body are affected. Pain is not usually a prominent symptom, even at the beginning of an attack. The anæsthesia is very characteristic. It is usually complete, but it is especially to be recognized by its abrupt termination in a line or a very narrow zone which extends entirely around the body. The bladder and rectum are completely paralyzed.

Hæmatomyelitis.—In some cases of hemorrhage into the cord the sudden paraplegia has been preceded by evidences of subacute myelitis, such as fever, formications, partial or complete paralysis of the bladder, girdle sensations, spasms, or muscular twitchings. To these cases the name of hæmatomyelitis has been given, but they are to be viewed as instances of spinal apoplexy occurring in a myelitis. Softening of the cord and other evidences of inflammation may be found after death in the neighborhood of the clot, even when there have been no distinct symptoms of myelitis before the hemorrhage. The natural explanation of these cases is that the inflammation of the cord was provoked by the hemorrhage.

Hemorrhage into Spinal Membranes.—In other cases of very acute paraplegia, instead of the paralysis being abrupt, many minutes, or even hours, are required for its complete development, and during this time there is very great pain. Under these circumstances the lesion is a hemorrhage outside of the cord into the spinal membranes. The rate of the development of the paralysis

varies according to the amount and rapidity of the hemorrhage. The loss of power is due not to an immediate lesion of the cord, but to pressure upon the cord, and especially upon the motor-nerve roots, by the exuded blood. Unless the blood be in great amount and thrown out with excessive rapidity, the paralysis will grow more and more marked during several hours, and will ascend higher and higher. As the blood creeps up the spinal cord or forces its way downward, it tears the membranes away from the cord, presses or stretches or perhaps tears the posterior, as well as the anterior, spinal roots, and produces by this irritation of the sensory nerve-roots shooting, tearing, or burning pains, with more or less marked loss of sensibility in the affected parts. The anæsthesia is usually not as complete or as abrupt as in cases of intraspinal apoplexy. Nevertheless, if the clot be a large one the sensory palsy may be complete, and the zone between the anæsthetic and the sensitive portions may be very narrow. The bladder and rectum are very frequently paralyzed. Priapism or other evidences of genito-urinary irritation might naturally be expected, but I have never in my own cases seen them.

Paraplegias of rapid or slow, but not of immediate, development, are best studied under two headings: first, those which are accompanied with excessive pain; second, those in which there is no pain, or, at most, only moderate suffering.

Painful Paraplegia.—In the so-called painful paraplegia the suffering is usually intense, is often worse at night, and, although it may be persistent, is at least in its exacerbations paroxysmal. The pains, which are described as stabbing, lightning-like, burning, etc., take almost every conceivable form. They are frequently felt in the neighborhood of the rectum or along the urethra. Motility is generally very slowly lost. The paralysis may be accompanied by spasm, but almost invariably at last the muscles are relaxed. The knee-jerks, at first in many cases exaggerated, are finally abolished. Hyperæsthesia may exist in the beginning, but at last gives place to anæsthesia. Trophic changes usually come on early, and may be complete.

In painful paraplegia the lesion is either a disease of the lower vertebræ or else a growth, usually sarcomatous or distinctly cancerous, so situated as to involve the nerves in their emergence

from the sacrum. When the vertebræ themselves are affected, the disease is almost invariably cancerous. An aneurism by pressure upon the lower vertebræ may destroy them, and as the nerves become implicated the pressure produces symptoms somewhat resembling those caused by malignant growths.

Non-Painful Paraplegia.—A paraplegia without excessive pain may be developed in the course of from one to six days: such cases constitute a group sufficiently marked to be studied together, and to be subdivided into several sub-groups.

Of these, sub-group the first includes those cases which so nearly correspond to those described by Landry that they may be known as Landry's palsy, or ascending paralysis.

Ascending Paralysis.—In some of the cases of ascending paralysis the symptoms are preceded by evidences of nervous disturbance, such as feelings of weakness, irregular formications, spots of numbness, weariness and discomfort, and possibly occasional spasmodic contractions. Either with or without these prodromes great weakness of the lower extremities comes on, and increases until, in the course of a few hours, standing or walking is impossible.

The palsy usually appears first in the muscles of the foot, then in the legs, then in the thighs, until the whole leg is flaccid and without power. The symptoms steadily progress upward, involving soon the arms and finally the muscles of respiration, and in this way producing death. The temperature of the body is very rarely, if ever, above normal; but, according to Hammond, the affected limbs are distinctly lower in temperature than normal. The knee-jerk is in most cases diminished, and is not rarely in the latter stages of the attack abolished, but early in the paralysis, and even when voluntary motion is profoundly affected, it may be well preserved: neither the bladder nor the rectum is usually paralyzed.

According to Landry, who first gave the name of ascending paralysis to cases of this character, the order in which the muscles are affected by the paralysis is,—

First. The muscles which move the toes and foot, then the posterior muscles of the thigh and pelvis, and lastly the anterior and internal muscles of the thigh.

Second. The muscles which move the fingers, those which move

the hand, and the arm upon the scapula, and lastly the muscles which move the forearm upon the arm.

Third. The muscles of the trunk.

Fourth. The muscles of respiration, then those of the tongue, pharynx, and œsophagus.

In many cases the paralysis does not follow the course laid down by Landry; it is often more irregular, one arm or one leg being more paralyzed than the other; and cases are affirmed to have existed in which the symptoms began at the upper portion of the cord and ran rapidly downward, involving therefore the upper extremities first. It is stated by Levi (*Archives Gén. de Méd.*, sixth series, vol. i., 1885, 129) that Cuvier died from an acute descending paralysis, affecting the medulla almost in the beginning of the attack.

There is no pain during the whole attack, or at least nothing beyond discomfort, formications, or more or less distinct numbness. Usually cutaneous sensibility is not entirely destroyed; sometimes it appears to be but little affected; but in a few cases there has been almost complete anæsthesia. There are usually no trophic changes, so that bed-sores, if they ever occur, are very rare. In a few cases of acute paraplegia of doubtful character perforating ulcers have appeared. Œdema of the skin was noted by Eisenlohr, and in some cases there has been a profuse secretion of sweat. In a case under my own care it was found by staining the nails with nitric acid that there was a partial arrest of growth, which was much more marked in the most completely paralyzed portions of the body.

A very important distinction which divides the cases of acute paraplegia just spoken of is that in some the symptoms progress slowly, requiring several days for their full development, whilst in others the symptoms rapidly increase. It is uncertain whether there is a vital difference in the pathology of these cases, but clinically they differ, in that the symptoms when slowly developed are prone to be arrested, so that the patient escapes for the time being, and in some instances entirely recovers. On the other hand, when the palsy rapidly rises up the body during the first twelve hours, it is rarely arrested,—the patient usually dying in a few days of asphyxia from respiratory palsy.

A much more infrequent form of acute paraplegia than that

just described, and which is perhaps worthy of constituting a second sub-group of cases, is typified by a case reported by C. Eisenlohr, in which, after exposure, a man was taken with pains in his limbs, followed by a rapidly-ascending paralysis, which became so complete that he could not move either his hands or his feet. There was fever, exaggerated knee-jerk, œdema of the extremities, preservation of the normal electro-contractility of the muscles, and, after a few days, rapid recovery (*Archiv f. Psychiatrie*, Bd. v., 219).

Acute Central Myelitis.—In the second group of cases of acute paraplegia symptoms somewhat similar to those of ascending palsy are present, but the following important differences are well marked: the anæsthesia is much more pronounced, and may be complete; paralysis of the bladder and rectum occurs early; the reflexes are soon abolished, and trophic changes take place almost at once in the paralyzed muscles, so that in the course of a very short time faradic contractility is lost and the reactions of degeneration appear; trophic changes in other than muscular tissue also occur early; sloughing bed-sores, especially in the buttocks and heels, soon appear, and rapidly increase; œdema of the paralyzed parts occurs. Death in these cases may take place, as in acute ascending palsy, from implication of the muscles of respiration, but usually the patient dies from exhaustion, due in part to bed-sores.*

This form of acute paraplegia is clearly separated from the other varieties by the rapid trophic changes. It constitutes the so-called acute central myelitis, an affection in which the central gray matter of the spinal cord is attacked: the ganglionic cells swell up, lose their processes, become granular in the interior, are converted into shapeless round masses, and finally disappear entirely; whilst, at the same time, disintegration occurs in the tissue around them. (For cases, see Ross, *Diseases of the Nervous System*, also Wigglesworth, *Liverpool Med. Journ.*, July, 1885.)

Lesions of Ascending Palsy.—In our first group of cases of acute paraplegia, the so-called ascending or Landry's palsy, a large number of post-mortem examinations have been made by

* These cases will be more fully described in the next great group of paraplegias. See page 64.

trustworthy observers, and the spinal cord has been examined most thoroughly, without any lesion being detected. We must, therefore, conclude that in the majority of these cases in which death occurs in a few days no lesions can be found in the spinal cord. In some cases, however, of ascending paralysis the white matter of the spinal cord has been found greatly altered. (See my own case, *Therapeutic Gazette*, 1885, also case reported by Hoffmann, *Archiv für Psychiatrie*, 1884, p. 140.) In these cases of degeneration of the white matter the symptoms vary somewhat according to the tract of white matter which is especially attacked. The absence of muscular alteration is very well accounted for by the lack of change in the trophic ganglionic cells. Those cases of ascending paralysis in which no lesion of the spinal cord has been found may be theoretically accounted for by supposing that time has not elapsed for changes sufficiently gross to be recognized by the microscope to be produced. I do not think, however, that this can be accepted as a sufficient explanation of all the cases. It is probable that sometimes the symptoms are the result of a rheumatic* or other toxæmic arrest of function in the spinal cord, whilst I still believe that engorgement of the vast plexus of veins in the vertebral canal outside of the cord may cause an ascending paralysis. It is probably these cases of congestion that constitute the clinical group of ascending paralyses, in which the symptoms fail to reach a fatal issue and recovery occurs.

There are cases of ascending paralysis in which the symptoms seem to be half-way between those of group No. 1 and group No. 2. These may be explained as cases of organic disease in which the lesion attacks both the white and the gray matter,—the character of the symptoms varying as the white matter of the cord or the gray bears the brunt of the attack.

It must also be remembered that in most of the cases of ascending paralysis in which no lesion has been found the nerves were not examined, and that an inflammation or degeneration of the motor nerve may have existed in some of these cases; especially as Déjerine and Goetz have found in a case in which there was no demonstrable lesion in the spinal cord, changes in the anterior

* It seems to me very probable that such cases as those of Eisenlohr are rheumatic.

nerve-roots similar to those of parenchymatous inflammation or degenerative atrophy,—changes which appear to be very similar in character to those which I found in the white matter of a cord in a case of Landry's paralysis.

Multiple Neuritis.—There is a group of cases in which paraplegia may develop very rapidly and deepen into general palsy, with symptoms simulating those of true ascending paralysis, in which the lesion is an inflammation of the nerve-trunks. As much can be accomplished by treatment, it is very important to recognize the true nature of *multiple neuritis*. The disease may take a very acute form, with death in the course of a few days from paralysis of muscles essential to life, or it may run a prolonged subacute course. When recovery follows either the acute or the subacute form, more or less permanent structural change of the muscles may be left. In cases of a subacute type, instead of the symptoms simulating those of ascending paralysis, various disconnected portions of the body may be affected, and a true multiple palsy, or a single or a double monoplegia, be produced. Certain cases of violent sciatica with rapid loss of power in the leg afford instances of monoplegia from inflammation of a nerve-trunk, and I have seen a severe subacute multiple neuritis, with all the characteristic symptoms confined to the arms, producing a double brachial monoplegia. The clinical evidence shows that any nerve of the body or any combinations of nerves may be affected by a neuritis.

In a very large proportion of the cases acute general multiple neuritis has followed excessive exposure, and not rarely the exposure has been associated with extraordinary physical exertion. In many cases the disease seems to be rheumatic. Leyden asserts that the salicylates act remarkably well,—a statement which I can confirm from experience. Ordinary sciatica is very closely related to multiple neuritis. Usually, but not always, in sciatica, as in other forms of neuritis, the sensory filaments of the nerve are chiefly attacked; but it will be shown later that it is probable that inflammation of the motor filaments of nerves may occur without the afferent or sensory fibres being distinctly influenced.

Although multiple neuritis is often of rheumatic origin, it is not invariably so. It is especially frequent in persons who use alcohol in excess, and not rarely follows various infectious diseases

of a low type. The so-called diphtheritic paralysis is due to a multiple neuritis. The loss of power which may follow typhoid fever (see page 43) has, at least in some cases, similar origin. Lowenfeld has seen multiple neuritis after recurrrent fever and after erysipelas, whilst Déjerine has noted it in syphilitics, and its presence in the Japanese disease beriberi has been fully established.

Malignant Multiple Neuritis.—In the acute forms of multiple neuritis the constitutional symptoms may be very severe, and marked by great prostration and high fever. The onset is often very sudden, but may be more gradual. The loss of power may be pronounced within twenty-four hours. It usually begins and develops symmetrically in two, and sometimes in all four, of the extremities; but perhaps in the majority of cases the arms are the more urgently attacked. The muscles of the hands and forearms and those of the leg proper are the first to lose their power. Rapidly, however, the palsy creeps towards the trunk, and passes (in some cases) to the muscles of the face. Double vision may develop as the result of paralysis of the eye-muscles. Speech and swallowing become involved, and as the respiratory muscles fail in power the patient sinks into a fatal asphyxia.

The heart may be affected, and degeneration of its nerves and muscular fibres has been detected. The sudden syncopal deaths which sometimes occur after diphtheria probably have this origin. Sometimes preceding and sometimes immediately following the motor symptoms great disturbances of sensation appear. Violent rheumatic-like pains occur in the limbs, or fulgurant agonies shoot up and down the nerve-trunks. Formications,—a peculiar feeling of numbness,—all forms of paræsthesiæ,—may be present. Hyperæsthesia often accompanies the first stages of the disorder, but in advancing cases it is followed by anæsthesia, which may be a true *anæsthesia dolorosa*. (See section on Anæsthesia.) An almost pathognomonic symptom is anæsthesia of the skin, associated with excessive sensitiveness of the muscles and other structures reached by deep pressure. All kinds of sensation seem to be affected: not only does the patient fail to recognize the points of the æsthesiometer, but scratching, or finally even pinching, produces no pain, and heat and cold elicit no response. Dreschfeld and Leyden have noted a very pronounced girdle sensation, which

the first-named observer, with probable accuracy, believes to be the result of the inflammation of the trunkal nerves. The trigeminal nerve usually escapes; but two cases in which it was attacked have been reported by Lowenfeld (*Neurol. Centralb.*, 1885, p. 140). Pain on movement may be present, soreness of the muscles is often noted, but marked tenderness of the nerve-trunks upon pressure is pathognomonic. In a few cases the nerves of special senses have been attacked, and partial blindness and deafness have been noticed.

In marked cases of acute multiple neuritis the trophic changes are pronounced. In the course of a very few days, or even hours, the affected muscles begin to waste. Brenner and Bernhardt state that there is usually lowering of the electro-excitability of the muscles, and not a qualitative alteration; but cases have been reported in which there have been true reactions of degeneration. (See chapter on Trophic Changes.) The paralyzed muscle is flaccid and soft, the absence of contractures is characteristic, but in very old cases deformities from contractures of the normal antagonistic muscles may be present. Even late contractures of the paralyzed muscles are usually held to indicate the presence of some secondary disorder of the nerve-centres; but this seems to me doubtful. (See G. H. Roger, *L'Encephalé*, 1885, p. 140.) Trophic changes in other than the muscular tissues are not rare, such as pigmentation and thickening of the skin, eczematous eruptions, alterations of the nails, œdema, bed-sores, etc. A peculiar lividity of the affected extremities, due to vaso-motor weakness, has been much commented on.

Very early in acute multiple neuritis the knee-jerk disappears, but the superficial skin reflexes, such as that of tickling, may first be exaggerated, and for a long time remain fully as active as normal; finally, however, they too diminish. The sphincters rarely are attacked, although Leyden reports one case in which the bladder was affected.

The diagnosis of cases of multiple neuritis conforming to the type just detailed is easy. The combination of paralysis with atrophy of the muscle, excessive disturbances of sensation, and tenderness of the nerve-trunks marks the disorder at once. On the other hand, the recognition of some of the subacute forms of multiple neuritis is difficult.

The paralysis of acute multiple neuritis is caused by a number of distinct lesions, and is therefore a true multiple palsy, although it may simulate a paraplegia or a general palsy. Not rarely, however, the neuritis is limited to certain nerve-regions, as the brachial plexus, or even to a single nerve. In this way monoplegia and local palsies are produced. The course of such an affection may be very rapid, but usually it is subacute. Although this subacute multiple neuritis ought perhaps to be considered under the heading of Multiple Palsies, for convenience I shall discuss it here.

Subacute Multiple Neuritis.—The symptoms of the subacute cases of multiple neuritis may differ from those of the acute disease only in being more confined in their seat and less rapid in their course. In other cases the symptoms are in the subacute affections very different from those of the acute disorder. Thus, Leyden, Lowenfeld, and others have noticed as a first symptom in some cases a loss of co-ordinating power, producing a well-marked ataxic gait. Whenever in any case of multiple palsy there are marked disturbances of sensation and tenderness of the nerve-trunk on pressure, the diagnosis is sufficiently made out, even though the trophic changes come very slowly.

Diagnosis of Multiple Neuritis.—Although the diagnosis of a typical multiple neuritis is very easy, it is otherwise with aberrant forms. Thus, in the case recorded by H. Mieth (*Deutsche Med. Wochen.*, 1885) there was paralysis, with diminished reflexes and great difficulty of swallowing and of speech, without trophic changes in the muscles or in the tissues, and without distinct disturbances of sensation. It is well known that sciatica may occur without pronounced palsy or rapid wasting of the muscles, evidence that the sensory nerve-filaments may be attacked without serious involvement of the motor fibres, and it is probable that in cases like that reported by Mieth the motor filaments of the nerves are affected without the sensory being distinctly implicated. Desnos and Joffroy, indeed (quoted by G. H. Roger, *L'Encephalé*, 1885, 140), have proved, by post-mortem examination of a case where there had been no distinct disturbances of sensation, that this non-sensory multiple neuritis does occur. As Bernhardt says, the diagnosis between this affection and poliomyelitis becomes excessively difficult.

The matter is made more troublesome by the fact (cases, Pitres, *Archiv de Neurol.*, Bd. vi., 180) that in locomotor ataxia, in acute myelitis, and probably in all spinal diseases, the peripheral nerves occasionally undergo acute degeneration. There is, therefore, a descending as well as an ascending neuritis; and whenever there is found at an autopsy a degeneration both of the spinal cord and of the nerve-trunks, the question arises whether the original lesion was peripheral and centripetal or centric and centrifugal. In very few cases of the ascending palsy have the peripheral nerves been properly studied. Indeed, it is probable that the lesion of the cord which was found in the case reported by Dr. Dercum and myself was the result of an overlooked ascending neuritis.

It is not probable that a multiple neuritis can exist without the sensory filaments being at least so far implicated as to cause tenderness of the nerve-trunks, and in the absence of positive clinical evidence we must consider that the diagnosis between a multiple neuritis and a poliomyelitis can be made by pressing upon the nerve-trunks. It is possible, however, that the future may reveal the existence of a pure motor neuritis, and it is exceedingly important that observers should note in doubtful cases whether the nerve-trunks are or are not tender. Literature should not be further encumbered with reports of cases without autopsies, or with imperfect autopsies.

Reactions of Degeneration in Neuritis.—It has been stated by various observers that true reactions of degeneration are never to be obtained in the atrophied muscles of multiple neuritis; but this is undoubtedly an error, as I know from my own experience, which is confirmed by Dr. L. Lowenfeld (*Ueber Multiple Neuritis*, Munich, 1885) and by Professor Lancereaux (*Union Méd.*, July, 1885). Without doubt, however, as Bernhardt (*Verhandl. Int. Congr.*, 1884) states, the changes of electrical reactions come on less rapidly in multiple neuritis than they do in poliomyelitis. Professor Remak considers that the localization of the palsy is of diagnostic importance, between the two diseases; but in this he is not borne out by the clinical records.

Alcoholic Spinal Paralysis.—Under the head of alcoholic spinal paralysis Dr. H. Broadbent (*Medico-Chir. Trans.*, vol. lxvii.) has reported a form of disease which is probably alcoholic

multiple neuritis. Indeed, the abuse of alcohol seems to be a very important etiologieal faetor in multiple neuritis. Cases similar to those of Broadbent have been reported by Lancereaux and others. They vary eonsiderably in their features. There is at first gradually-increasing weakness of the lower extremities, when suddenly marked loss of power beeomes manifest in the extensor muscles of the forearm, giving rise to double wrist-drop. The flexors of the hand may be affected very early. Usually, somewhat later they become paralyzed, so that the hand is like a flail. Although the patient can walk and the movements of the elbow and shoulder are vigorous, the paralysis rapidly advances, until all four extremities are almost eompletely motionless, the arms, as a rule, being more seriously implicated than the legs. The reflexes are abolished. There is usually no pain, though the museles may be tender on handling, and the sphineters retain their funetional power. In one of Dr. Broadbent's cases, however, sharp pains shot down the legs, and there was incontinence of urine. In the course of a very few days the muscles of the trunk become implicated, and the patient dies of paralysis of respiration, precisely as in ascending palsy. Loss of tone in the eapillaries, with consequent livid congestion of dependent parts, is said to be diagnostic of this form of paralysis. Careful examination of the spinal cord failed to detect any lesions. The cases are said to be much more frequent in women than in men.

Subacute Paraplegia.

The second group of organie paraplegias eomprise those eases in whieh the paralysis develops so slowly as to require many days or weeks, but not months, for the symptoms to become very pronouneed. In this group are ineluded cases of transverse myelitis and of general myelitis. It must be remembered that in exceptional cases either of these affections may develop with phenomenal rapidity and give origin to an acute paraplegia.

Transverse Myelitis.—Transverse or compression myelitis is invariably the result of disease of the vertebræ or of the membranes of the cord. The most frequent cause is scrofulous or syphilitic degeneration of the vertebræ; but syphilitic, cancerous, or other tumors may produee the disease.

In the majority of cases local pains precede the development of those symptoms which are directly due to the myelitis. The seat of these pains varies according to the seat of the disease,—the rule being that the pains are in the distribution of those nerves whose roots pass through the inflamed or degenerated vertebral tissues. Along with these symptoms of irritation of the posterior spinal roots there may be cramps or convulsive movements, evidences that anterior or motor nerve-roots are implicated: except in very rare instances, these motor symptoms are much less marked than is the sensory disturbance.

When the cause of a transverse myelitis is cancer, the pain is generally atrocious, radiates in all directions along the trunks of the nerves, is described as shooting and tearing and burning, and is usually associated with very pronounced hyperæsthesia of the skin. The pangs themselves are paroxysmal in violence, although present, to some extent, the whole time. The crises of the paroxysms are said by Grasset to occur almost invariably at night. They are increased by any attempt at motion, and seem at times to be brought on by slight touches. Hyperæsthesia in these cases is often finally replaced by anæsthesia without the pains being relieved, and herpetic eruptions appear along the courses of various nerves. In its acute form this affection is the painful paraplegia, or paraplegia dolorosa, already described (see page 50). Painful paraplegia may also be due to cancerous tumors outside of the spinal column, so situated in the neighborhood of the sacrum as to involve the numerous nerves coming from the cauda equina. The pain may precede the palsy by a considerable time. I have known it to be the first symptom of pelvic cancer.

The symptoms directly due to the transverse myelitis itself are most marked in regard to motion. Voluntary power is finally lost; the reflexes are exaggerated, but not to the degree seen in cases of general myelitis, except when secondary spinal lesions have followed upon the transverse myelitis.

In the latter stages of transverse myelitis there is always paraplegia, but in the beginning, if the inflammation be more intense on one side of the spinal cord than on the other, one extremity may be more profoundly affected than another, and a brachial or crural monoplegia or even an apparent hemiplegia may be present.

Close inspection will, however, almost always detect some weakness of the leg at first thought to be intact. When the transverse myelitis is high up in the cord, the arms may be affected before the legs.

Sensibility in transverse myelitis at first is dulled, but finally it is completely lost. Paralysis of the bladder and rectum occurs, and in many cases the symptoms finally are not to be distinguished from those of a more general subacute myelitis. Indeed, a subacute myelitis may very well be developed out of a transverse myelitis.

Early Diagnosis of Transverse Myelitis.—It is a matter of the greatest importance to recognize early the presence of vertebral inflammation, especially when the latter is of a scrofulous character. By paying attention to the pain this can frequently be done before the occurrence of a transverse myelitis, or the appearance of any striking local evidences of disease of the vertebræ. This is especially true when the disease is located in the upper dorsal or the lower cervical region. Whenever a patient with the aspect of a person suffering from severe disease has, without obvious cause, an intense fixed pain about the shoulders or in the arms, it should be borne in mind that this pain may be due to irritation of nerve-roots caused by incipient disease of the vertebræ or of the spinal membranes. When other causes for such pain can be excluded, and deep pressure over the spine produces pain, or pain can be caused by jars of the spinal column, or by blows upon the top of the head directed downward, the diagnosis is sufficiently probable for therapeutic purposes. Some aid may be gained by the application of *Rosenthal's test*, upon which, however, in my experience, not much reliance can be placed. The test is made by placing one pole of a faradic battery in contact with the front of the body, and passing the other pole down the centre of the vertebral column, when, if any inflammatory lesion exists, pain will be developed at the seat of the change. Unfortunately, tenderness may often be found when there is no vertebral disease, and, if the anterior portions of the vertebræ alone be implicated, pain may not be elicited by the current.

Care may at times be necessary not to confound incipient vertebral disease with the so-called *spinal anæmia* or *spinal irritation* (see article on Pain); but the aspect, mode of talking, and general

conduct of the patients are so different in the two diseases that the experienced physician can hardly be misled, although these differences may be very hard to put in words. Moreover, in spinal irritation there is intense tenderness to the slightest touch, whilst in the early stages of true vertebral disease tenderness is evoked only by firm pressure. Further, the distant pains of spinal irritation lack the fixedness and intensity so characteristic of vertebral disease.

It must be borne in mind that a syphilitic or other *growth* springing *from the membranes* of the cord may involve the nerve-roots, and may produce transverse myelitis, with early symptoms very like those of vertebral caries. Deep vertebral tenderness under these circumstances develops only late in the disease, when the bodies of the vertebræ are affected, although hyperæsthesia over the vertebral column, as over other parts of the body, may be caused by a secondary neuritis.

Cervical Pachymeningitis.—A class of rare cases which are especially liable to be confounded in their first stages with incipient Pott's disease, and in their latter stages with organic disease of the spinal cord itself, is contained in cervical pachymeningitis, an affection whose history was especially elaborated by Charcot and Joffroy. This disease is, indeed, in its latter stages accompanied by a transverse myelitis, and many of the symptoms which are at such time present are due to the transverse myelitis. In the first stage there is vague pain in the neck and in the occipital region of the head, which is exaggerated by pressure and movements and is associated with more or less marked spasm of the muscles of the neck. Frequently there are paroxysms of pain associated with temporary torticollis. These symptoms slowly increase, often for from two to five or six months, until, at last, the suffering is very great, especially at night, and in irregular paroxysms, whilst immobility of the neck from spasm becomes complete. Radiating pangs shoot along the nerves of the neck into the arms, back, and head, and hyperæsthesia and fulgurating pains often occur in the hands. In some cases the pain in the neck is not at all marked, whilst the peripheral pains are excessively violent. Digestive disturbance and vomiting are not rare. As the disease increases, paralysis appears usually first in the arms; sometimes, however, it is distinctly paraplegic in type, and in some cases even hemiplegic.

Subacute Central Myelitis.—Among the cases of subacute paraplegias belong many of the cases of central myelitis. In this disease, with or without prodromes, the patient is taken with a fever, which may be preceded by a chill and be accompanied by much sweating. Pains of greater or less severity are felt in the legs and in the lumbar regions. These rapidly increase, and may take a girdle form. Power is also rapidly lost in the legs, but at the same time spasmodic jerkings and irregular muscular contractions are developed. In very rapid cases in the course of two or three days the paraplegia may be almost complete, and death has occurred as early as the fifth day. The reflexes are exaggerated, but before death may be weakened and finally abolished. Paralysis of the bladder early appears. The urine has to be drawn off by a catheter, and is strongly ammoniacal. Large eschars now form upon the buttocks, or upon the heels where they rest upon the bed. These rapidly increase, and add greatly to the exhaustion of the patient. Sensibility, usually less early affected than motion, soon, however, becomes blunted, and at last there may be complete anæsthesia. The symptoms which have just been mentioned constitute a typical case of a general acute myelitis, and may be developed so rapidly as to put the case in the group of acute paraplegias.

In some instances of myelitis the evidences of motor and sensory excitement are more marked than has been indicated, the irregular muscular contractions are associated with an almost tetanic rigidity of the muscles of the trunk and of the limbs, and along with the pain there is marked hyperæsthesia. If the myelitis attack the upper dorsal region, the symptoms in the arms may be even more pronounced than those in the legs, and cough, with marked dyspnœa, may be prominent. Gastric crises with violent vomiting may be very distressing, and may simulate in their severity those of locomotor ataxia. The difficulty of swallowing is often very great. If the cervical region be affected, irregular dilatation or contraction of the pupils may be produced; and a very prominent symptom in some cases has been an extraordinary reduction of the pulse, which has been noted to fall as low as 28 per minute.

CHRONIC PARAPLEGIA.

Chronic paraplegia (group three of my arrangement) includes those cases in which many weeks, months, or years are required for the full development of the symptoms. In arriving at a diagnosis in these cases, sensation, the condition of the bladder, and the state of the muscular system are especially to be studied.

There are two diseases which are common causes of chronic paraplegia. This does not include cases of multiple palsy, which, as already stated, when located especially in the lower legs, may give rise to an apparent paraplegia, and may pursue a chronic course. The nature of these cases can almost always be recognized by the irregularities in the development of the palsy, and by the wasting of the affected muscles, conjoined with the absence of the symptoms of chronic general myelitis. (See page 58.)

Both in chronic myelitis and in sclerosis the symptoms vary according to the region of the cord attacked; but in sclerosis with motor paralysis distinct disturbances of sensation, as well as paralysis of the bladder or of the rectum, are extremely rare.

Chronic Myelitis.—In chronic myelitis anæsthesia is present only in exceptional cases, and still more rarely is it preceded by hyperæsthesia. Very seldom is there severe pain, although formications and paræsthesia are frequent. When the disturbances of sensation are marked, the whole of the structure of the cord must be considered as implicated. Trophic changes may occur in the muscles and other tissues, but it is remarkable how frequently the trophic cells of the gray matter escape, even when all the rest of the cord is attacked. In the beginning of the disease the reflexes may be increased, to be finally abolished.

There is a form of chronic myelitis in which the reflexes remain exaggerated for many months, and in some of these cases they are grossly exaggerated, and a certain amount of tonic spasmodic contraction exists in the affected muscle; but the rigidity does not compare with that which is seen in the sclerotic form of paraplegia. On the other hand, irregular spontaneous jerkings of the leg, spasmodic twitchings of the muscles, and painful contractions at night are more frequent in myelitis than in sclerosis.

Girdle Sensation.—A very important symptom which is present

in both forms of chronic paraplegia is the girdle sensation, usually felt in the abdomen, but in some cases very distinct at a lower or higher position in the legs. Its seat is probably connected with the position of the disease in the spinal cord.

In chronic myelitis the patient walks slowly, dragging the feet with evident effort. The posture does not essentially differ from the norm, there are rarely bizarre movements of the legs, and any tottering or unsteadiness is evidently from feebleness.

Spasmodic Tabes.—When, in a case of chronic paraplegia developed during adult life, the muscles of the leg are exceedingly rigid, firmly contracted, with their tendons projecting and hard, and the legs and feet are more or less distorted, the patient is suffering from spasmodic tabes. In the early stages of this affection the patients complain that they are readily fatigued, and that their gait is dragging. Even before there is distinct loss of power the patient will be troubled at night, especially after a hard day's march, with clonic or tonic spasms, which cause the legs to stiffen suddenly or to be jerked about. A little later the stiffness and loss of power combine to produce a very characteristic gait. The contractures of the various muscles prevent the bending of the joints of the knee and hip, whilst the great power of the muscles of the calf tends to draw the heel up and to thrust the toe downward. Consequently, the foot can be lifted sufficiently from the ground to make a step only by raising and rotating the pelvis so that the body is inclined towards the leg upon which the patient rests during the step, whilst the moving foot is slowly thrust forward. The toes appear to stick to the ground, and are only with the greatest difficulty sufficiently raised to be pushed forward. The steps are of necessity very short,—it may be only three or four inches. As the leg is put forward, not rarely violent tremblings affect it, and in some cases these movements are so rhythmical as to throw the heels of the patient up and down in regular vibrations. As the disease progresses, the contractures of the muscles of the calf become so great that the heels are permanently drawn from the ground, and the patient rests upon the toes. Under these circumstances the trunk is of necessity thrown forward, and is preserved from falling only by means of crutches or canes held well in advance of the body. A little later than this all power of locomotion is lost, and not

rarely the patient is confined to bed, or, if he attempts to sit, must be propped up in a chair, with his feet supported in front of him.

When the power of locomotion is lost, the leg is usually flexed upon the thigh, the heel drawn up, and the toes turned inward, these positions being due to the superior power of the posterior muscles of the thigh and leg and of the abductor muscles as compared with their antagonists. In some cases the patient lies with the legs stiffly extended, very rigid, the feet inverted and often crossed. The bladder and rectum are not affected. In most cases of spasmodic tabes the muscles do not undergo wasting, and their electrical reactions are not altered in quality, and are even more sensitive than normal. The spinal lesion in spasmodic tabes is sclerosis of the lateral columns, usually known as *lateral sclerosis.*

Amyotrophic Lateral Sclerosis.—In certain cases of spasmodic tabes the muscles are very much wasted, and may finally disappear (see chapter on Trophic Changes), although their electrical reactions are exceedingly slow to alter. These are instances of the so-called amyotrophic lateral sclerosis, in which the lateral columns and the large multipolar cells of the adjacent gray matter are simultaneously diseased. Frequently in these cases the wasting of the various muscles is irregular and unequal, so that the case presents the aspect of a multiple palsy attacking a person already affected with disease of the lateral column; but in some instances the trophic changes develop *pari passu* with the paralysis.

Multiple Spinal Sclerosis.—When the symptoms of spasmodic tabes are associated with more or less irregularly developed disturbances of sensation similar to those of locomotor ataxia, disseminated sclerosis will be found after death; that is, patches of sclerosis, irregular in size, shape, and seat, are scattered through the cord. The symptoms in such cases vary as the lesions predominate in one or other of the spinal tracts.

MONOPLEGIA.

A monoplegia is a paralysis of one extremity. When of the arm, it is spoken of as brachial monoplegia; when of the leg, as crural monoplegia.

Hysterical Monoplegia.—Hysterical monoplegia is rare, but

may occur, and is not infrequently attributed to a real or an alleged injury. Under such circumstances the true nature of the loss of power is apt to be overlooked. If contractures come on immediately after a real or an alleged injury, the paralysis is probably hysterical; but complete relaxation may exist in an hysterical monoplegia. When after a traumatism the paralysis and the relaxation are complete and there is no wasting of the muscles, the affection is usually hysterical, since in all cases of total or nearly total loss of power from injuries to a nerve the muscles rapidly change. Irregularities in the anatomical relations between the disturbances of sensibility and the alterations of mobility indicate an hysterical origin, but these relations may, in hysteria, conform to the organic type.

Irregularity of Implication of Muscles.—An organic monoplegia may be looked upon as a collection of local palsies: thus, in a case of centric brachial monoplegia (see page 72) the cortical nerve-centres which preside over the various groups of muscles in the arm are located so near to one another that they are involved in a common, wide-reaching clot or other lesion; and in a peripheral brachial monoplegia many distinct nerve-trunks or many distinct groups of spinal ganglionic cells are involved. It is owing to these facts that the intensity of the palsy in brachial monoplegia varies in the different groups of arm-muscles according as one centre or the other is more implicated in the lesion.

Double Monoplegia.—Two monoplegias may coexist in the same patient, and, when they happen to be on the same side of the body, may simulate a hemiplegia. The distinction, however, between such a double monoplegia and a hemiplegia may be vital. In a double monoplegia there are necessarily two lesions, whilst in a hemiplegia the paralysis is the result of a single lesion. When an enormous clot upon the brain-surface affects the whole motor zone, it gives rise to a paralysis which must be considered a hemiplegia.

A monoplegia may be due to a lesion of a nerve, or of the large multipolar cells in the anterior cornua of the spinal cord, or of the brain-cortex. In making the diagnosis in any case the general situation of the lesion is first to be determined.

Varieties of Monoplegia.—There is nothing in the paralyzed muscles which distinguishes a palsy due to a disease of the ante-

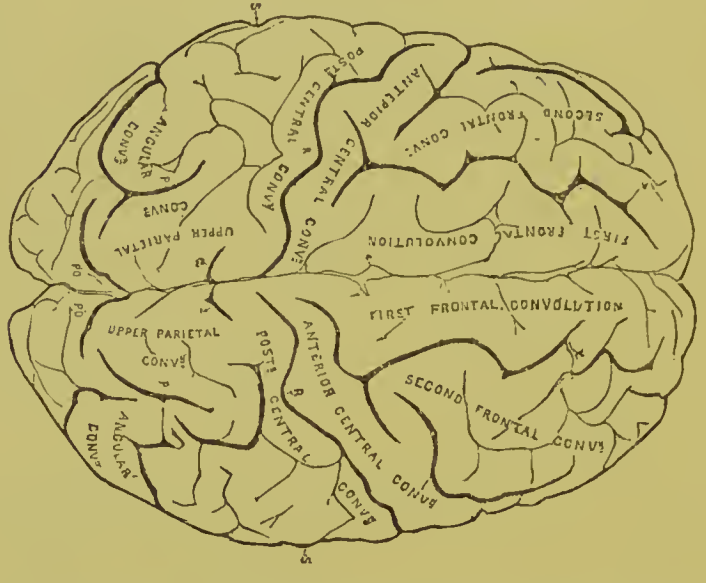
rior spinal cornua from one which is the result of an affection of the nerves. These two classes of cases are for the purposes of diagnostic discussion profitably grouped together as peripheral palsies. Paralyses due to lesions of the brain-cortex are readily distinguished from peripheral palsies by a study of the affected muscles. Cerebral paralyses, therefore, constitute a second group of monoplegias, which may be spoken of as centric palsies. It must, however, be remembered that a nerve-trunk may be paralyzed by a lesion within the cranium and yet the palsy belong to the peripheral group. Thus, if by an organic change in the pons or in the medulla, or by an exudation or a tumor at the base of the brain, the integrity of one of the cranial nerves be interfered with, the results are palsy and structural changes identical with those which would follow section of such nerve after its emergence from the cranium.

An acute peripheral paralysis is always to be distinguished from a centric palsy by the rapid occurrence of structural changes in affected muscles,—changes the nature of which will be fully discussed in the chapter on Trophic Alterations, to which the reader is referred for details. Suffice it for the present to state that in centric palsy the muscle does not undergo change, whilst in peripheral palsies the muscle in three or four days after the inception of the lesion begins to lose its power of responding to a rapidly-interrupted faradic current, and in the course of a week or two sensibly wastes.

Anatomy of the Cortex.—In order to understand the production and diagnosis of cerebral monoplegias it is necessary to pay attention to the following considerations. The ganglionic cells of the brain-cortex, which originate the impulses that call forth voluntary movements, are scattered over a considerable portion of the cortex, so that a lesion may very readily affect some of the cells without influencing others. The nerve-fibres which pass downward from these cortical centres are, however, gathered together into a fasciculus so small that a lesion affecting one portion of the fibres almost invariably exerts a greater or less influence upon the remainder. For these reasons centric or brain monoplegias are of cortical origin. It frequently happens in a hemiplegia that the arm or the leg is more affected than is its co-sufferer, because the clot presses more severely upon one portion

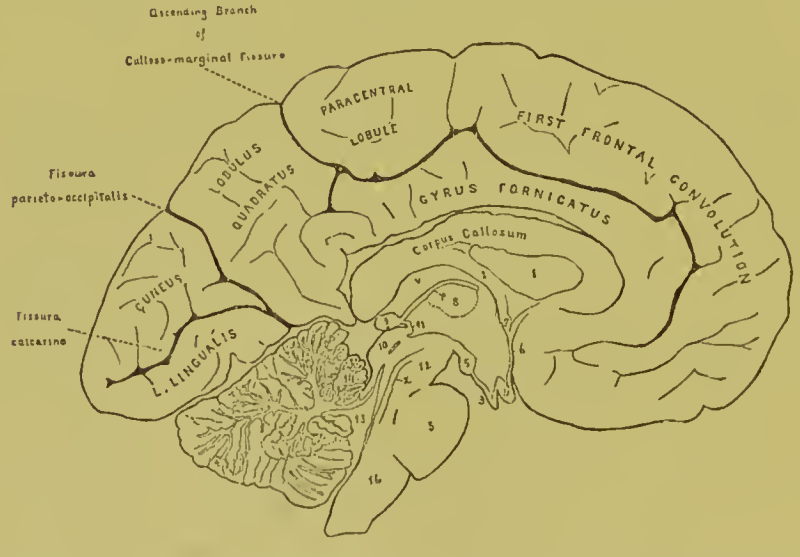
of the conducting fasciculus in the brain than upon another. It is, however, excessively rare for a single set of fibres of this fasciculus to be affected by itself, and a monoplegia to be thus produced.

Fig. 3.



For the purposes of diagnosis the cortical structure of the cerebrum may be divided into three portions. That which in diagram No. 3 lies in front of the letter A, Fig. 5, comprising the

Fig. 4.

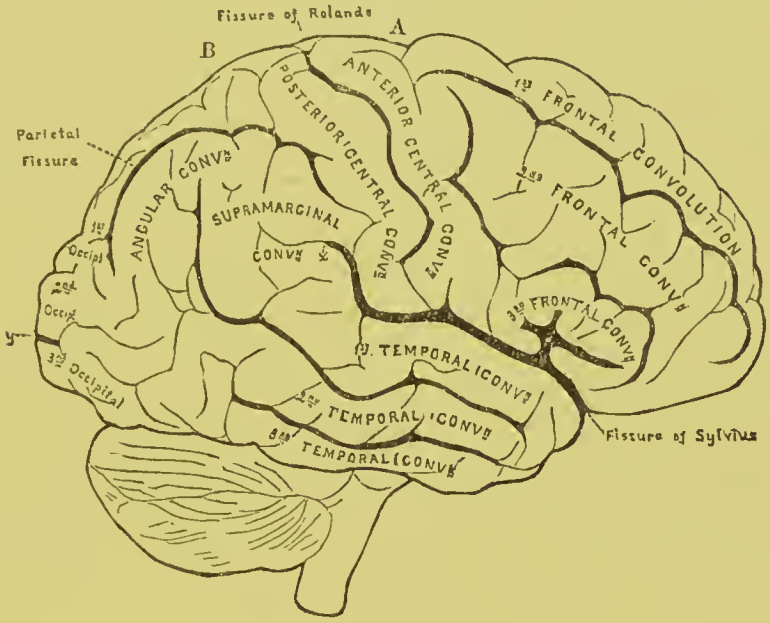


so-called frontal convolutions, is the *inert zone*. Extensive lesions may exist in this portion of the brain and produce no symptoms whatever. The region between letters A and B comprises the

motor zone of the brain, extending as far downward as the fissure of Sylvius. The cortical region posterior to the letter B may be considered as chiefly sensory in its function.

The motor zone of the cerebral cortex is composed of the anterior central convolution (ascending frontal convolution), the

FIG. 5.



posterior central convolution (ascending parietal convolution), and the paracentral lobule, and appears in some cases to reach into the lobulus quadratus, the supra-marginal convolution, and even the gyrus fornicatus. According to the collection of cases made by Exner, the extent of this zone is usually greater in the left than in the right hemisphere. The cortical cells, which are situated in the motor zone, appear to be more or less imperfectly grouped together, so that those muscles which are anatomically closely related to one another and habitually act together receive their impulse from contiguous cortical cells.

Various attempts have been made to isolate and locate these groups by the study of the recorded cases in which the symptoms have been observed during life and the lesion after death. There appears to be a certain amount of uniformity in the position of the arm-, leg-, and face-centres, but this uniformity is not rarely departed from. It is very clear that the groups overlap one another. It may be asserted, as a general rule, that the anterior central convolution is more active as a motor centre than is the

posterior central convolution, and that the cells which are connected with the lower extremities are situated in the upper portion of these convolutions, with the arm-centres below them and the centres connected with the face chiefly located in the anterior central convolution close to its foot.

Exner arrives at the conclusion that the cortical arm-centres occupy the paracentral lobule, the anterior central convolution with the exception of its lowest portion, and the upper half of the posterior central convolution, reaching, in rare cases, into the lobulus quadratus and the gyrus fornicatus. The field of the lower extremity he locates in the paracentral lobule, the upper third of the anterior central convolution, and some portions of the upper third of the posterior central convolution. In rare cases, this field seems also to enter the lobulus quadratus (especially in the left hemisphere, in which it may even reach the cuneus).

The facial nerve zone, according to Exner, occupies the lower half of the anterior central convolution and the lower third of the posterior central convolution. The centre for the tongue is in the foot of the anterior central convolution, although there is at least one case on record in which a lesion in the supra-marginal convolution produced hypoglossal symptoms.

Cerebral Monoplegia.

Abrupt Cerebral Monoplegia.—Sudden monoplegia of cerebral origin may be due to traumatisms, to hemorrhage, and to arrest of the circulation by thrombi or embolisms. There are no known symptoms which enable us to decide whether the cause of a sudden brain-monoplegia is a hemorrhage or an arrest of circulation.

Progressive Cerebral Monoplegia.—A cerebral monoplegia may be developed suddenly or gradually. When it comes on slowly it is due to a progressive lesion situated in the motor zone of the cortex. Such a lesion is of the nature of a tumor, of an outgrowth from the skull, or of a localized meningitis with much exudation or great disturbance of the circulation. It must be remembered that the cortical layer of the brain is supplied by blood-vessels which pass from the membranes into the brain-substance, and are, in the cortex, terminal arteries which do not

anastomose. Any change in the membranes may, by producing pressure upon the blood-vessels, interfere so seriously with the circulation in the cortical substance of the brain as to cause progressive degeneration resulting in loss of function without there being a direct propagation of disease from the membranes to the brain-substance. In syphilitic meningitis the lesion has a very distinct tendency to invade the contiguous brain-substance. Syphilitic disease also much more frequently locates itself in the motor zone than do benign tumors: consequently, in a very large proportion of the cases of progressive cerebral monoplegia the lesion is a local syphilitic meningitis.

Peripheral Monoplegia.

Monoplegia from Pressure on Nerve.—Very frequently a man will awaken from a drunken stupor to find that his arm is paralyzed; and it is common for a young bridegroom to get up in the morning with his arm in a similar condition. In either case pressure of a head upon the arm has been the cause of the trouble. The loss of motor power in these cases may be more or less complete; there is usually tingling and a distinct feeling of deadness, but pronounced anæsthesia is very rare, although it may occur. The musculo-spiral nerve, on account of the manner in which it winds around the arm, is especially apt to suffer, and, as it chiefly supplies the extensors of the forearm, these muscles are usually most severely affected. A not rare form of pressure-palsy is the so-called *crutch-palsy*, in which a double or single monoplegia is produced by the pressure of the crutches or crutch upon the nerves in the axilla. Pressure-palsy in most cases yields readily to treatment, and is rarely, if ever, sufficient to produce trophic changes.

Monoplegia from Injury or Disease of Nerves.—Complete palsy of the arm may result from injuries to the brachial plexus, as well as from multiple neuritis or other idiopathic diseases of the same, and from affections of the cervical spinal cord which involve the nerve-roots. Under the latter circumstances the monoplegia is almost always double. If the functional power of the brachial plexus be abolished, the muscles will rapidly waste and die. I have seen a condition of the brachial plexus allied to

cerebral and spinal concussion, caused by an ocean wave striking from above downward on the supra-clavicular region and followed by total loss of nerve-function. Traumatic and idiopathic diseases of the sciatic nerve may cause a crural monoplegia.

Toxic Monoplegias.—Various metals are capable of producing monoplegias, but, practically, lead is the only one which frequently causes such palsy. Plumbic monoplegia is usually brachial, and resembles most closely that form of organic monoplegia which is due to pressure upon the nerves. It is to be separated from this, usually at a glance, by being double; but I have seen one case of single brachial monoplegia due to lead, and one case of pressure-monoplegia in which both arms were affected on account of the patient's work requiring him to labor with outstretched arms resting near the body upon a narrow board. In the plumbic single monoplegia the paralysis was caused by the local absorption of the metallic salt, in whose solution the hand and arm were habitually immersed. Under such circumstances, the diagnosis must be made out by careful attention to the history. In plumbic monoplegia the extensors are almost solely affected; but this may also be true of the paralysis produced by pressure, since the musculo-spiral nerve may be alone implicated, and it chiefly supplies the extensors of the hand. It is affirmed that in plumbic paralysis the short extensor of the thumb escapes, and that this is pathognomonic.

MULTIPLE PALSY.

A multiple palsy is one in which two or more groups of dissociated muscles are involved. The symptoms vary according to the seat and nature of the lesion, and are, in a word, the associated symptoms of the various local palsies of which the multiple palsy is composed.

A multiple palsy may be of cerebral or of peripheral origin. The nature of any individual case is to be determined in the same way as in monoplegia, and the reader is referred to page 69 for the distinction. There is, however, one form of peripheral multiple palsy in which, although the muscle wastes, the reactions of degeneration do not appear. (See Progressive Muscular Atrophy.)

Cerebral Multiple Palsies.

The cells in the cerebral cortex which are connected with motion are, as has already been explained, so placed that those which lie near one another affect associated or closely-related muscles. Cerebral multiple palsies are therefore rare, because a small lesion of the cerebral cortex affecting contiguous cells produces a monoplegia, whilst a lesion sufficiently large to affect the whole motor zone of one hemisphere produces a hemiplegia. A multiple cerebral paralysis can be produced only by two lesions, or by a lesion of such character that it affects scattered areas of the cortex. A clot or an embolus affects almost invariably a definite area of the cortex, either large or small, and produces, therefore, a hemiplegia or a monoplegia. Consequently acute cerebral multiple palsies are among the most infrequent of diseases.

Syphilitic Multiple Palsies.—Syphilis is especially apt to produce two or more lesions in the brain: consequently a progressive cerebral multiple palsy is in the adult usually syphilitic, due to the action of two or more slowly-developing patches of gummatous meningitis.

Multiple Cerebral Sclerosis.—Occasionally the disease known as multiple cerebral sclerosis affects the cortical region, or its minute scattered patches may even invade the interior portion of the brain in such a way as to produce multiple palsies. The diagnosis of this disease is especially to be made upon the existence of tremors, which occur in the affected parts only during voluntary movement. The symptoms and course of the affection will be discussed in detail under the head of Tremors.

Spastic Infantile Paralysis.—One of the most frequent of the progressive or chronic multiple paralyses of cerebral origin is the affection which is known as spastic paralysis of childhood. This is a disease or condition which, beginning in early childhood, continues through life. The palsy may take the form of a hemiplegia, paraplegia, monoplegia, or multiple palsy, and in some cases the paralyzed region is so small that the patient might be considered to be suffering from a local paralysis. The name of spastic infantile palsy which has been given to this disease expresses well its characteristic features,—namely, the presence of contractures and the age of attack.

The contractures are so severe as to give rise to very pronounced distortions. In the hand the fingers are partially flexed and irregularly drawn apart, whilst the palm is usually somewhat cup-shaped, and the wrist and even the elbow may be in a perpetual flexion. In the feet almost any variety of pes equinus may occur, but most commonly the toes are drawn downward and inward, and the sole is somewhat inverted; not rarely the feet are crossed.

Not only may the muscles of the extremities be attacked, but also those of the trunk; and more frequently the neck is affected, so that the head is held in various bizarre positions. The muscles of the face are rarely paralyzed, but permanent grimaces and even various squints may show that the muscles of the head have not escaped. The rigidity is usually not so complete but that by means of moderate force an imperfect return of the limb to its normal position may be temporarily produced.

Spontaneous movements in the limbs are rare, but there may be a single or double athetosis and even true choreic movements. It is especially in this disease that the so-called "*associated movements*" first described by Westphal occur. When this condition is present, movements made in the non-paralyzed extremity are similarly but imperfectly executed in the paralyzed part. Thus, in a right-sided hemiplegia, when the fingers of the left hand are opened or shut those of the right hand follow the impulse. As stated by Westphal, these associated movements do occur in the clot-hemiplegia of adults, although they are very rare.

In spastic infantile paralysis partial loss of power is frequent, the muscles responding slowly and imperfectly to the will, but without choreic jactations. In other cases the affected muscles, although unable to respond properly to the will, are thrown by its efforts into more or less irregular and varied choreiform contractions.

The muscles themselves are wasted, firm in substance, with distinctly tense tendons, but never offer any electrical reactions of degeneration, and, indeed, preserve fully their relations with the electric current. The reflexes are never lessened; usually they are somewhat exaggerated, and in some cases they are very markedly increased, so that the condition known as *spinal epilepsy*, in which violent general contractions are produced by slight external irritation, may exist. The presence of these grossly exag-

gerated reflexes or of spinal epilepsy in any individual case seems, however, to show that the spastic palsy is not of the pure type, but that there are associated with it, either as a result or as an accompaniment, sclerotic changes in the spinal cord especially affecting the lateral columns. As already stated, no distinct trophic changes occur in the muscles, neither are true trophic changes to be found in the other tissues, but there is very frequently a partial arrest of development of the affected limb, so that not only is it less in diameter but also in length, and the bones and joints are unduly small.

There is no disturbance of sensation.

Epilepsy is common, and often severe. In the earlier stages of the disease the convulsions usually begin in the affected limbs, and they may for a time even be completely monoplegic or unilateral, but sooner or later they become universal. Not rarely the attacks of major epilepsy are associated from time to time with pronounced petit mal.

In most cases there is a lack of mental power. A partial or complete aphasia is frequent, and every grade of imbecility, from idiocy to the nearly normal condition, may be found. Often also there is marked asymmetry of the skull; or the head may be excessively large, or perhaps more frequently abnormally small.

Almost invariably spastic infantile palsy dates back to very early childhood, although occasional cases are developed as late as the tenth or twelfth year. I have seen a number of cases in which the symptoms were noticed a very short time after a birth which had been instrumental, and I believe that not infrequently the disease is due to a meningeal hemorrhage caused by the injury to the child's head by the forceps of the accoucheur. In another set of cases the attack dates to convulsions occurring during early childhood. These cases afford a twofold history. In some of them there is no apparent cause for the convulsions. The child is seized with unconsciousness accompanied with violent convulsive movements, and is left paralyzed. Under these circumstances the convulsions have been invariably more severe in the subsequently paralyzed limb. I believe that the pathology of these cases is that an apoplexy has occurred with a clot, and this clot has been the cause of the convulsive seizure and of the after-palsy. In the second set of cases the convulsions result from

an obvious cause, such as gastric irritation or the coming on of a scarlet fever or other exanthematous disease. If under these circumstances the child emerges with a permanently paralyzed limb, the convulsion has probably produced a rupture of the blood-vessel in the brain and consequent hemorrhage. It would appear, however, that it is possible for a convulsion occurring in a child to be accompanied with so much cerebral congestion of a local character as to produce a slight temporary loss of power in the limb without the presence of a clot in the brain; and when this congestion is frequently repeated, a progressive structural change of the affected portion of the brain is set up. At least such is the explanation of cases like those described by Jules Simon (*Revue mens. des Maladies de l'Enfance*, December, 1883), in which the child passes through a series of more or less distinct convulsions, each followed by evidences of local weakness and a little stiffness, disappearing after a few hours, but continually growing more pronounced and more permanent after the successive fits, until finally a true contracture is produced.

Lesion of Spastic Paralysis.—Infantile spastic paralysis is the result of sclerotic and atrophic changes in the brain. Such changes in most cases date back to the occurrence of the hemorrhage, but in other instances, and especially in the slowly-developed cases last spoken of, they are the result of frequently-repeated irritative congestions of the brain-substance. The seat of these lesions varies indefinitely, as does the position of the resultant palsies and the degradation of the mental condition of the child.

The only disease with which spastic infantile paralysis could be confounded is lateral spinal sclerosis and multiple sclerosis of the brain or cord, which certainly may exist in children as in adults. In spinal lateral sclerosis there are no convulsions and no arrest of mental development, whilst the reflexes are grossly exaggerated. In multiple spinal sclerosis the symptoms resemble those of lateral sclerosis, but are less severe, and usually pains or other evidences of sensory disorder mark the presence of sclerotic patches in the sensory region of the cord. In multiple cerebro-spinal sclerosis the peculiar tremors are present. It must, however, be remembered that, especially in childhood, cortical brain sclerosis is very prone to give rise to, or to be associated with, secondary conditions of the spinal cord, and that not rarely cases occur in which the

symptoms of these three so-called diseases are mingled because the sclerotic changes are so widely scattered through the nervous system.

Peripheral Multiple Palsies.

Peripheral multiple palsies may be acute or chronic. An acute or subacute organic peripheral multiple paralysis is due either to a lesion of the nerves or to an affection of the ganglionic cells of the anterior cornua of the gray matter of the cord, called poliomyelitis. The diagnosis as to which of these parts is affected may be difficult. The history of the case is of great importance. In the absence of traumatism the paralysis is almost invariably due either to poliomyelitis or to multiple neuritis. The diagnosis of multiple neuritis has been fully discussed under the head of Ascending Palsy. The reader is, however, reminded that the important points are the presence of pain and tenderness over the nerve-trunks in neuritis, and their absence in poliomyelitis; also that in poliomyelitis the trophic changes occur much more rapidly and completely than in neuritis.

Diphtheritic Paralysis.—A very important form of peripheral multiple paralysis is that which follows diphtheria. The symptoms usually commence in two or three weeks after the inception of the disease, but may come on as early as the sixth day, or may be delayed for four weeks or even longer. In the majority of cases the original attack of diphtheria has been mild, and in the lower classes I have on several occasions seen the primary disease entirely overlooked. In its typical form the palsy begins first in the palatine muscles, and is usually revealed by a peculiar twang of the voice, resembling that commonly spoken of as nasal. At this time, or shortly afterwards, there is a difficulty in swallowing, which may become so severe that all liquids are returned by the nose, and even the power of swallowing solids is almost lost. If the palate be at this time examined, it will be noticed to be flabby, motionless, and more or less anæsthetic. The laryngeal muscles may now be attacked and difficulty of respiration be experienced from paralysis of the abductors of the vocal cords. When the laryngeal symptoms are severe, the nasal voice is replaced by a whisper, or by a total extinction of sound. The tongue, the lips, and even the muscles of mastication may be af-

fected, so that the patient is unable to chew food or to retain the saliva, which constantly dribbles from the mouth. The eye-muscles are usually the first affected after those of the throat. Paralysis of accommodation comes on early, and is soon accompanied by loss of power of some of the muscles of the eyeball, giving rise to strabismus and diplopia. Complete mydriasis, with ptosis, may also occur from loss of power in the oculo-motor nerve. About this time weakness of the legs is noticed and rapidly deepens into a more or less complete paraplegia. The arms also, and even the muscles of the trunk, may be affected, so that in some cases a general palsy results. Even the muscles of organic life may be implicated, as is shown by obstinate constipation from paralysis of the intestinal walls, paralytic retention of urine, and cardiac failure. The circulation in severe cases is feeble, and the surface cold and blue. In the majority of cases diphtheritic paralysis is recovered from; but death may result—from choking, produced by the slipping of a piece of food into the larynx; from inanition, the effect of the loss of power of taking food; from asphyxia, due to paralysis of the respiratory muscles; or from cardiac failure, which may develop with great suddenness. In some cases paralysis of the diaphragm is revealed by the epigastrium and hypochondrium being drawn inward instead of being curved outward during inspiration. This condition is one of grave danger. When it occurs, or when the pulse is exceedingly weak, or the first sound of the heart is markedly diminished, absolute quiet is of the utmost importance, as any exertion may produce sudden death from paralytic asphyxia or syncope. Sensory disturbances always accompany diphtheritic palsy. They consist of anæsthesia rather than of pain, though numbness and tingling, with formications, are occasionally felt. The distribution of the anæsthesia varies greatly. It may be almost universal, or may be confined to the throat, and in some cases it affects other mucous membranes, so that defecation and micturition may be performed without conscious feeling. Only in rare cases are the nerves of special sense affected.

Poliomyelitis.—The diagnosis of a fully-developed poliomyelitis is rarely obscure, but in very acute cases the nature of the attack is in its beginning often overlooked. Not infrequently the symptoms develop with comparative slowness, weakness of the part affected being the first evidence of disease. On the other

hand, in some cases there is during the earlier days of the attack great systemic disturbance, with high fever and an array of symptoms sufficiently resembling those of malarial or typhoid fever to be readily mistaken for them. An examination of the legs and arms under these circumstances will detect the presence of some localized or wide-spread loss of power, and thereby reveal the nature of the disease. Unless the practitioner habitually examines the muscles of the extremities and trunk in cases of obscure fever occurring in children, he will be very apt to make a mistake in the early diagnosis of acute poliomyelitis. The general course and symptoms of acute poliomyelitis will be discussed in the chapter on Trophic Changes. At present I shall only further point out certain affections which may be confounded with the disease.

Local Atrophies.—Care is sometimes necessary not to confound with a poliomyelitis certain atrophies of the muscles which follow injuries or are associated with surgical inflammations. Very common among these are *palsy* of the *deltoid*, the result of a fall upon the shoulders. Paralysis and atrophy of other muscles from a direct blow may happen, but on account of its situation the deltoid is much more frequently affected. After the muscle has more or less completely recovered from the immediate effect of the bruise, it is found to be completely palsied. I believe that in such a case the loss of power is due to concussion, or even more severe injury, of the peripheral nerve-endings in the muscle. After fractures and luxations, palsies of the neighboring muscles, with atrophy, are prone to occur. In some cases they are the result of a direct injury to the nerve, or of pressure upon the nerve by callus, etc. In other cases no such immediate lesion can be made out. Even when a nerve-trunk has been injured it may often be noted that the paralysis and atrophy are not confined to the muscles innervated by the nerve, and they may become so general and wide-spread as to involve all the muscles of the extremity. More rarely the muscles upon the opposite side of the body are affected with the same changes. To the atrophies in the neighborhood of the lesion the name of Atrophy by Propagation has been given, whilst the changes at a distance are sometimes spoken of as Reflex Atrophies. The lesion in these cases is probably an ascending neuritis.

Joint Atrophies.—John Hunter was the first to call attention to the muscular atrophies which follow disease of the joints, and in 1845 Bonnet discussed the question at length, and showed that chronic synovial inflammations may produce wasting not only in the immediate neighborhood of the affected articulation, but also throughout the limb, and that the affected muscles are much paler than normal and lose a portion of their fibres. Most frequently in cases of arthritis the extensors are the first to be profoundly affected. This atrophy is not simply loss of muscle-tone from want of use. It is always accompanied by paralysis, and sometimes the paralysis comes first. It may continue long after the arthritis has been cured. It is usually, but not always, in proportion to the inflammation in the joint, and is more frequent with blennorrhagic and scrofulous inflammations than with rheumatic arthritis. M. Gosselin has called attention to the peculiar atrophy with contracture of the long peroneal muscle, which is not rarely seen in medio-tarsal arthritis. The loss of opposition to antagonistic muscles sometimes in these cases produces great distortion in the foot and hand.

Toxæmic Peripheral Palsies.—An affection which is liable to be confounded with idiopathic poliomyelitis, and in which the lesion is probably degeneration of the ganglionic spinal cells, is produced by poisonous doses of lead or of arsenic, and probably of other metals. The loss of power sometimes affects almost the whole muscular system, but may be irregularly located either in the upper or in the lower extremities. According to my experience, almost complete paraplegia, with comparatively trifling arm-paralysis, is most frequent after arsenical poisoning, whilst in lead-poisoning the upper extremities, and especially the deltoid muscles, are prone to be attacked. It must be remembered, however, that in either of the poisonings any associated or scattered groups of muscles may be affected. The symptoms develop rapidly, the affected muscles wasting and presenting in a very short time the electrical reactions of degeneration, and finally, in extreme cases, passing into a condition in which they fail entirely to react to electric currents. The symptoms are similar to those of ordinary cases of acute poliomyelitis. The toxic nature of the affection is indicated by the disease occurring in adults, in whom acute poliomyelitis is excessively rare, by the rapidity of the changes, by the

wide distribution of the paralysis, and sometimes by the loss of power over the bladder and rectum. The diagnosis is further to be made out by the history of the case, or, if this be wanting, by the occurrence of disorders of sensation as well as of motion. Widespread or narrowly-limited spots of anæsthesia can usually be discovered, at least in the early stages of the disorder. Not infrequently the anæsthesia finally disappears, although the motor paralysis remains. Especially in arsenical poisoning, and in the early stages of the attack, violent pains shoot through the limbs, following more or less distinctly nerve-trunks, and giving rise to the suspicion that the pathology of some of these cases is a peripheral neuritis. It is especially important that a correct diagnosis be made of these cases, because they are nearly always curable by treatment. The occurrence of an acute poliomyelitis in the adult should always create in the mind of the practitioner a strong suspicion of a toxic origin, and the urine should be examined for lead and arsenic. Finding the metal of course decides the diagnosis, although very frequently the paralysis outlasts the apparent elimination of the poison, and no metal can be detected in the urine. In lead-poisoning, the occurrence of the blue line upon the gums is decisive; but its absence is not proof of the absence of lead from the system.

Chronic Peripheral Palsy.—The most frequent form of chronic peripheral paralysis occurring in adults is that which is known as *progressive muscular atrophy*. In this disease, without an obvious cause, wasting, with loss of power, appears in the affected muscle, and progresses so slowly that years may be required for the destruction of the part. Under these circumstances the loss of power is in direct proportion to the wasting of the muscle, but normal electrical reactions of the wasted muscles persist until almost the last fibre is destroyed. The explanation of this characteristic symptom will be given in the chapter on Trophic Changes, where, also, the course of the disease will be spoken of in detail. The lesion is a progressive degeneration of the ganglionic motor cells of the spinal cord, and hence the term chronic poliomyelitis which is sometimes employed as the name of the disease. Progressive muscular atrophy is to be recognized by the slowness of its course, the peculiarly irregular groupings of the palsies, the fibrillary contractions and wasting of the muscles, with conservation of

their normal electrical reactions, and the absence of pain and of paralysis of the bowels or of the bladder.

Pseudo-Hypertrophic Paralysis.—A disease which causes multiple palsies, and therefore requires mention here, although probably not an affection of the nervous system, is pseudo-hypertrophic paralysis. During infancy or early childhood it is noticed that the child is very easily fatigued, walks unsteadily, falls frequently, and continually supports himself by clinging to chairs or other furniture. He soon begins to go up-stairs with difficulty, drags himself along by the balusters, and finally cannot ascend except on his hands and knees. He is also affected when walking on the level ground: the feet are held widely apart; the gait becomes straddling. In stepping, the active foot is raised from the ground by an elevation of the pelvis, and the trunk is bent towards the passive leg. Lordosis is apparent, both in standing and in walking. The abdomen is thrust forward and the shoulders backward, so that a vertical line dropped from between the shoulders passes behind the sacrum. In the advanced stages the child is unable to rise from the floor or the chair in the ordinary manner. He drags himself up with his hands; or, if he be lying down, and no support be forthcoming, he gets upon his hands and knees, and then, grasping each thigh alternately, is able to raise himself sufficiently from the floor to get first one and then the other foot upon its sole. He then lays hold of his thighs with successive grasps, one above the other, and thus, as it were, climbs up them to a standing position. This method of getting on the feet is pathognomonic of pseudo-hypertrophic paralysis. A test which, in the young child, is almost diagnostic, even at a time when the changes of the muscles are not visible to the eye, is the inability of the standing child to raise himself upon his toes.

Sooner or later in pseudo-hypertrophic paralysis the affected muscles become larger and firmer, with abnormally rounded outlines. Occasionally some of them undergo atrophy. The order of development of the paralysis is usually first in the calves, then in the gluteal muscles, then in the muscles of the back, then in those of the thigh, and finally in those of the arms. This progression is not, however, invariable. The electro-contractility of the muscles may for a time be normal: later it is diminished to the faradic current, abnormal or increased to the galvanic current: finally,

it is lost to both currents. These alterations are, however, developed very slowly. In the advanced stages the knee-jerk is abolished. There is no paralysis of the bladder or of the rectum, and no disorder of sensation. Characteristic changes will be seen in fragments of muscles withdrawn by cutting trocars.

LOCAL PARALYSIS.

Local paralyses may be either centric or peripheral. In the large majority of cases they are peripheral. The diagnosis of the nature of a local palsy is to be made in exactly the same manner as with monoplegias and multiple palsies, and it does not seem necessary to repeat what has been already said.

In order to facilitate the recognition of the muscles and nerves which are affected in any individual case of paralysis, I propose to consider succinctly the paralyses which result from a loss of power in the various nerves and their tributary muscles.

Oculo-motor Paralysis.—Dilatation of the pupil, ptosis or dropping of the upper lid, paralysis of accommodation, squint with consequent double vision, are symptoms of loss of power of the oculo-motor nerve, whose superficial origin is from the inner border of the crus cerebri, the deep origin being in the locus niger of the peduncles and the gray nucleus in the floor of the aqueduct of Sylvius slightly below the tuberculæ quadragemini. Partial paralysis of this nerve is frequent. In such cases the symptoms vary according to the portion of the nerve affected. The functions of the eye-muscles are as follows: to turn the eye—superior oblique, downward and outward; inferior oblique, upward and outward; superior rectus, upward and inward; inferior rectus, downward and inward; internal rectus, directly inward; external rectus, directly outward. All these muscles are supplied by the oculo-motor nerve except the superior oblique and the external rectus. When one of these muscles is paralyzed a squint results. In order to determine which muscle is affected, it is only necessary, at least in cases of fresh paralysis, to note the position of the head. The rule is, the head is so deflected that *the chin is carried in a direction corresponding to the action of the paralyzed muscle.* Megalopsia, or macropsia, is a condition of vision in which objects look larger than normal. It is said to indicate paralysis of the external

rectus. Micropsia, in which objects look smaller than normal, is said to indicate paresis of the internal rectus muscle. These two symptoms are very rare. I have never seen either of them.

Oculo-motor palsy is in the majority of instances peripheral, due to pressure upon the nerve by basal exudations. The common cause in adults is a syphilitic meningitis, in children tubercular or rachitic meningitis; but it may be produced by a cancerous or benign tumor. For other details upon oculo-motor palsy as a symptom see page 41.

Fourth or Trochlear Nerve.—Loss of power of the superior oblique muscle of the eye is diagnosed by the fixedness of the eye when the head is moved, or, in other words, by the moving of the eye with the head. Double vision occurs whenever the subject attempts to look straight downward, or at objects situated towards the paralyzed side; but the second image disappears when the head is turned to look towards the sound side. The distortion of vision is especially manifested when any attempt is made to pick an object, as money, off a table. The nerve involved is the fourth, trochlear, or pathetic, whose apparent or superficial origin is in the superior peduncle of the cerebellum. Its fibres have been traced into the peduncle to the valve of Vieussens, near the tuberculæ quadragemini, where they decussate with corresponding filaments of the opposite side.

The fourth or trochlear nerve and the sixth or abducens, like the oculo-motor, are frequently paralyzed by basal meningitis, but loss of power in them may be due to centric lesion, such as a clot, a tumor, or a degeneration of the nucleus.

Fifth or Trigeminus Nerve.—Loss of power in the muscles of mastication, *i.e.*, the temporal, masseter, and pterygoids, and in the mylo-hyoid, digastric, tensor palati, and tensor tympani, indicates paralysis of the motor root of the fifth or trigeminus nerve. This root has its apparent origin in the side of the pons; its deep origin is in a nucleus just below the lateral angle of the fourth ventricle, immediately in front of the nucleus of the facial nerve.

Sixth or Abducens Nerve.—Paralysis of the abducens nerve causes loss of power in the external rectus, with consequent internal strabismus, or squint, double vision, and sometimes macropsia. Internal squint does not, however, always indicate

paralysis of the sixth nerve, because the weakness of the external rectus muscle is a very frequent result of imperfection of vision. The apparent origin of the abducens nerve is from a groove between the anterior pyramid of the medulla and the posterior border of the pons. There are usually two roots, one from the medulla and the other from the pons. The fibres have been traced to a nucleus which lies underneath the fasciculus teres in the floor of the fourth ventricle. A few fibres are believed to pass from this nucleus upward and across to join the third nerve of the opposite side. In this way are explained certain rare cases of conjugate paralysis involving the internal rectus of one side and the external rectus of the other side, and accompanied by atrophy of the nucleus of the abducens nerve.

Facial Nerve.—Of all the nerves of the body the facial or seventh nerve is most frequently paralyzed. The superficial origin of this nerve is in a groove between the olivary and restiform bodies of the medulla. Its deep origin is probably in the upper portion of the pons, although its fibres have not been distinctly traced farther than a nucleus in the upper half of the floor of the fourth ventricle near the postero-median fissure. It supplies all the muscles of the face, except those of mastication, also the levator palati and the tensor tympani.

Centric paralysis of the facial nerve is common. It is never complete, and almost invariably affects the muscles about the corner of the mouth. It is revealed by the slight drawing of the mouth to the opposite side, by loss of power of whistling or of fine articulation, and by a little flabbiness of the affected part. In deep coma this palsy can often be recognized by the peculiar puffing out of the corner of the mouth during expiration.

Peripheral palsy of the facial nerve is very frequent, constituting the affection sometimes known as *Bell's palsy*. The paralysis is always complete, or nearly so. The face is strongly drawn towards the opposite side. The power of completely closing the eye is lost, because the orbicular muscle is not able to raise the lower lid. The wrinkles in the forehead and the various folds of the skin, to which the face owes so much of its expression, entirely disappear or are greatly flattened out. The saliva is with difficulty retained. Articulation is distinctly impaired. During the process of chewing, the food is very apt to accumulate between

the teeth and the cheek, on account of the flaccidity of the buccinator muscle. Mastication is not otherwise interfered with, because the muscles of mastication are not supplied by the facial nerve. Bilateral facial paralysis, *facial diplegia*, or simultaneous palsy of both facial nerves, is exceedingly rare, though it is sometimes produced by a long transverse lesion crossing the anterior half of the pons, or by a similar transverse lesion encroaching upon the facial nerves after their emergence. It is characterized by a fixed, immovable, expressionless countenance, a peculiar dropping of the angles of the mouth and collapsed appearance of the nostrils during inspiration, and a marked flapping in and out of the cheeks during respiration. The voice is usually nasal, and the articulation very bad owing to an impossibility of pronouncing labial consonants. There is excessive difficulty in retaining the food between the teeth, and the saliva in the mouth.

There are three distinct positions at which lesions of the facial nerve-trunk may occur and produce characteristic symptoms. The first and most frequent is that in which the point of paralysis is at or immediately after the escape of the nerve from the temporal bone. Under these circumstances the paralysis is limited to the muscles of expression.

The second form of facial palsy is that in which the lesion is situated above the origin of the chorda tympani nerve, but on the distal side of the petrosal nerve. Under these circumstances to the paralysis of expression is added great diminution of the sense of taste in the anterior two-thirds of the tongue.

In the third variety, the lesion is behind the ganglionic enlargement which gives origin to the third petrosal nerve. There is now loss of power in the muscles of expression, loss of taste, paralysis of the soft palate, as revealed by a depression of the arch of the palate upon the affected side, and a loss of power in the tensor palati muscle, so that the soft palate is drawn towards the normal side. At the same time the sense of hearing is generally abnormally acute, and the secretions of the parotid and submaxillary gland are deficient.

Paralysis of the facial nerve may be due to tumors at the base of the brain, to disease of the petrous portion of the temporal bone, or to simple rheumatic neuritis. Owing to the exposed position of the nerve and the habitual nakedness of the face, this paralysis

is frequently produced by exposure to cold draughts or winds, especially after heating of the body. Paralysis from exposure usually involves only that portion of the nerve which is external to the bony canal, though sometimes the inflammation may extend backward into the canal. Complete peripheral palsy of the whole nerve is in the great majority of cases due to disease of the bone, or to tubercular or syphilitic basal meningitis.

Glosso-Pharyngeal Nerve.—Paralysis of the glosso-pharyngeal nerve is revealed by difficulty of swallowing, with great tendency to regurgitation of food through the nostrils, and the loss of taste in the posterior third of the tongue. The superficial origin of the nerve is in the groove between the lateral tract and the restiform body of the medulla oblongata. Its fibres have been traced to a nucleus in the floor of the fourth ventricle.

Spinal Accessory Nerve.—The spinal accessory nerve is composed of fibres springing from the lateral columns of the medulla oblongata and of fibres which rise between the anterior and posterior roots of the first and fifth cervical nerves, the two parts being united in the cranium and escaping as one nerve through the jugular foramen. The spinal accessory nerve sends communicating fibres to the pneumogastric, which appear to reach the laryngeal muscles, since in paralysis of the spinal accessory the voice becomes hoarse and unnatural; the act of deglutition is also somewhat affected. It affords the chief but not the only supply of the sterno-mastoid and trapezius muscles.

Sterno-mastoid Muscle.—Paralysis of the sterno-mastoid muscles causes slight elevation of the chin, with rotation towards the paralyzed side, causing an oblique position of the head. There is difficulty in depressing the head towards the paralyzed muscle, whose normal outline in the neck is also softened down. If both muscles be affected, the head is held straight, and is rotated with great difficulty; great difficulty is also experienced in depressing the chin.

Trapezius Muscle.—Paralysis of the trapezius muscle is shown by sinking of the point of the shoulder, drooping downward of the scapula, the inferior angle being in the relation of adduction to the spine as compared with its fellow, and prominence of the clavicle and supraclavicular space. If there is also difficulty in raising the scapula and clavicle, and in elevating the arm, the upper fibres

of the muscle are especially involved; while if the scapula is not easily approximated to the spinal column, the middle and lower fibres are chiefly affected. If after complete paralysis of the trapezius there is absolute inability to draw the scapula towards the spine, palsy of the *rhomboideus major* and *rhomboideus minor* muscles may be inferred. Under similar circumstances loss of the power of elevating the scapula, and of moving the neck after fixation of the scapula, indicates paralysis of the *levator angulæ scapulæ*.

Long Thoracic Nerve.—If the scapula is drawn upward with its lower angle approximated to the spine, and if during the act of elevating the arm the lower angle of the bone does not describe an arc outward, as it normally should do, but approaches still nearer to the spine, while the vertebral border stands out in a wing-like manner, leaving a well-marked depression between it and the thorax, then there is paralysis of the *serratus magnus*, which is supplied by the posterior thoracic or long thoracic or external respiratory nerve of Bell.

Subscapular Nerves.—Difficult adduction of the arm, with loss of the normal power of depressing it and drawing it backward, especially in the act of placing the hand in contact with the buttock, shows paralysis of the *latissimus dorsi* muscle, which is chiefly supplied by the subscapular nerves.

Inability to perform properly inward rotation of the humerus, diminished power of pronation, excessive outward rotation of the upper arm, and consequent faulty position of the hand, denote paralysis of the *subscapularis* and *teres major* muscles, which receive their nerve-supply from the subscapular nerves.

Supra-scapular and Circumflex Nerves.—Impaired power of outward rotation of the humerus, and consequent difficulty in performing such acts as writing, drawing, and especially sewing, in which this movement is essential, together with excessive inward rotation, even to the point of turning the ulnar border of the hand uppermost, indicate paralysis of the important external rotator of the humerus, the *infra-spinatus* muscle, as well as of its assistant, the *teres minor* muscle. The former is supplied by the supra-scapular nerve, and the latter by the circumflex.

When the arm cannot be directly elevated,—*i.e.*, brought at right angles with the trunk,—but hangs helpless close to the

thorax, and, later, when a definite space appears between the head of the humerus and the acromion, there is paralysis of the *deltoid muscle*, which is supplied by the circumflex nerve.

Anterior Thoracic Nerves.—Inability to adduct actively the arm so as to draw it across the chest, or to place the hand on the opposite shoulder, abnormal prominence of the ribs and intercostal spaces, and loss of tension of the anterior border of the axillary space, are the symptoms which show paralysis of the *pectoralis major* and *pectoralis minor* muscles, supplied by the anterior thoracic nerves.

Musculo-Cutaneous Nerves.—Absence of the greater part of the power to flex the forearm, with loss of some of the power of supination, and partial lack of ability to draw the humerus forward, inward, and towards the scapula, point to paralysis of the group of muscles supplied by the musculo-cutaneous nerve,—viz., the *biceps cubitis*, the *coraco-brachialis*, and part of the *brachialis anticus*.

Musculo-Spiral Nerve.—If the hand hangs at right angles to the forearm (wrist-drop) and the power of extension at the wrist-joint and elbow-joint is absent, with the hand in pronation, the fingers bent, and the thumb flexed and adducted, the deformity is characteristic of the group of muscles supplied by the musculo-spiral nerve and its posterior interosseous branch,—viz., the *triceps* and *anconeus*, the *supinator longus*, the *extensor carpi radialis longior* and *brevior*, and all the extensor muscles of the superficial and deep posterior brachial regions. Other prominent symptoms are that the effort at extension of the fingers is possible only in the second and end phalanges, while the first phalanges are more flexed (the interossei flexing the first phalanges and extending the others). The hand-grip is weakened unless the wrist-joint be put into extension, and when the hand and forearm are put prone upon the table there is diminished power of abduction and adduction. The forearm cannot be brought midway between pronation and supination, and when it is in this position the ability to perform elbow-joint flexion is impaired. Finally, the forearm cannot be extended upon the arm.

Median Nerve.—Loss of the power to flex all the second phalanges and the end phalanges of the index and middle fingers; preservation of this motion in the first phalanges (interossei), and

its partial preservation in the two outer fingers; inability to flex the thumb or bring it in apposition with the little finger; diminished power in flexing the wrist, which, when this is attempted, throws the hand into a marked adduction; and impaired pronation with lessened sensibility of the first two fingers and radial side of the ring finger, indicate paralysis of the median nerve. This nerve supplies all the *flexor* and *pronator* muscles of the deep and superficial anterior brachial region, with the exception of the *flexor carpi ulnaris* and the ulnar half of the *flexor profundus digitorum*, which are supplied by the ulnar nerve, and also all the muscles of the thumb except the *adductor* and one head of the *flexor brevis pollicis*, and finally the two outer *lumbricales*.

Ulnar Nerve.—Imperfect flexion of the hand, which is towards the radial side; impaired power of adduction of the hand; lessened ability to separate the fingers (abduction) or to bring them together (adduction); absence of the power to flex the first row of the phalanges and extend the last two rows; almost entire immobility of the little finger; difficulty in attempting to oppose the thumb to the metacarpal bone of the index finger, with disturbed sensation of the entire little finger and ulnar side of the ring finger, constitute the symptoms of paralysis of the muscles supplied by the *ulnar nerve*. These muscles are the *flexor carpi ulnaris*, part of the *flexor profundus digitorum*, the *interossei*, and the two inner *lumbricales*, all muscles of the little finger, and the *adductor* of the thumb and one head of the *flexor brevis pollicis*.

When the interossei and lumbricales are no longer able to flex the first row of the phalanges and extend the other two rows, but the extensor communis digitorum excessively extends the first row of the phalanges, while the flexor muscles bend the second and third row, the condition of "claw-hand" is produced, which may mean paralysis of the ulnar nerve just above the wrist, so that the innervation of the interossei and lumbricales alone is affected.

Spinal Nerves.—If the head hang forward and cannot be extended, or at least can be extended only by the aid of a swinging motion, there is paralysis of the extensors of the cervical vertebræ, —*i.e.*, the *rectus capitis posticus major* and *minor*, the upper portion of the *trapezius*, and the *splenii*.

When the spine tends to assume a posterior curvature, most marked in the dorsal region, and the patient presents the ap-

pearance of "old man's back," in which he cannot voluntarily straighten the curvature, although this may be done by passive action, there is paralysis of the *extensor muscles of the back*, chiefly the *longissimus dorsi* and *sacro-lumbalis*, and the condition of *paralytic cyphosis*. The production of *lateral curvature*, or *paralytic scoliosis*, means that the paralysis is limited to one side only.

When a patient carries the body with the upper portion bent backward, so as to throw it behind the centre of gravity, and when the body, if inclined too far anteriorly, falls forward and cannot again assume the erect posture until the hands, being placed upon the legs, help the arms by a sort of climbing process to bring the body again to its backward posture, the condition of paralysis of the extensor muscles of the lumbar region obtains,—*i.e.*, the *erector spinæ* and its divisions. In this condition the patient further stands with the head bent forward, walks with a swaying motion of the trunk, and when he sits down the upper part of the body apparently sinks, so that the dorsal spine is bent (cyphosis), while there is a deep concavity of the lumbar spine (lordosis). The nerves concerned in these palsies of the back are the posterior branches of the spinal nerves, cervical, dorsal, or lumbar, according to the region involved.

Ilio-Hypogastric—Ilio-Inguinal—Intercostal Nerves.—Inability to compress properly the contents of the abdominal cavity, so that such acts as urination, defecation, and vomiting are performed with difficulty, and diminished power in the effort of respiration, together with a tendency to fall backward when the upper part of the trunk is inclined posteriorly, show paralysis of the *abdominal muscles*, which are supplied by the ilio-inguinal, ilio-hypogastric, and lower intercostal nerves.

Anterior Crural Nerve.—Loss of the power to flex the thigh upon the abdomen and extend the leg at the knee, and impaired ability to raise the body from the recumbent posture, and to perform the acts of walking, running, going up-stairs, and the like, are the symptoms which indicate paralysis of the group of muscles supplied by the anterior crural nerve,—viz., the *iliacus*, *pectineus*, and *all the muscles on the anterior surface of the thigh* except the tensor vaginæ femoris.

Obturator Nerve.—When the act of pressing the knees firmly together, or of crossing one leg over the other, cannot be properly

performed, and when there is impaired power of external rotation of the thigh while in the sitting posture, the indications are that there is paralysis of the *gracilis* and *adductor* muscles of the internal *femoral region* and of the *external obturator* muscle, which group is supplied by the obturator nerve.

Superior and Inferior Gluteal Nerves.—Uncertainty in the act of walking or standing, together with absent power of internal rotation of the thigh and impaired power of external rotation; difficulty in abducting the thigh, with disturbed relation of the thigh to the pelvis, and inclination of the latter to the opposite side during attempted action on the part of the affected limb, are the symptoms which point to paralysis of the muscles supplied by the superior and inferior gluteal nerves. The inferior gluteal nerve is distributed to the *gluteus maximus*, which muscle can forcibly extend the thigh on the pelvis and perform outward rotation of the thigh. The superior gluteal nerve passes to the *tensor vaginæ femoris* and to the *gluteus medius* and *minimus*. The anterior fibres of these latter muscles rotate the thigh inward, while their posterior fibres rotate it outward. This group (gluteal), when taking their fixed point from the pelvis, are abductors of the thigh; when they take their fixed point from the femur, they support the pelvis on the femur. The tension of the fascia lata, which may be slackened in palsy, is usually maintained by the gluteus maximus and the tensor vaginæ femoris.

Sciatic Nerve.—Inability to flex or bend the knee, to oppose resistance to passive extension of the knee, and to raise the heel towards the buttock, would show loss of power in the *semimembranosus, semitendinosus,* and *biceps femoris* muscles, a group supplied by the great sciatic nerve. This is a possible form of paralysis; but more usual are the palsies which occur from affections of the principal branches of its distribution, and consist in loss of the extension and flexion of the foot and toes and abduction and adduction of the foot.

External Popliteal Nerve.—If the foot cannot be flexed or abducted, nor completely adducted, and hangs downward, so that the patient in the act of walking raises the foot by flexing the hip-joint and then places it again upon the floor in such a manner that the point of the toes and the outer border of the foot touch the ground first, the symptoms are characteristic of paralysis of

the muscles supplied by the external popliteal or peroneal nerve. This nerve, through its two branches, the anterior tibial and musculo-cutaneous, supplies the muscles of the anterior portion of the leg and the extensor brevis digitorum on the dorsum of the foot.

Internal Popliteal and Post-Tibial Nerves.—If the foot cannot be extended, nor the toes be flexed or moved laterally, and if the patient cannot stand upon his toes or properly adduct the foot and raise its inner border, paralysis of the group of muscles supplied by the internal popliteal nerve and its continuation, the posterior tibial nerve, may be inferred. This group consists of the muscles of the calf and of the deeper posterior leg-region, and, through the external and internal plantar nerves, of those of the sole of the foot. In this palsy the great toe can neither be flexed nor moved from side to side. The foot may assume an appearance similar to the "claw-hand" described under palsy of the ulnar nerve, and for the same reasons.

NOTE.—I desire to acknowledge the assistance derived from Dr. K. Heller's *Path. und Therap. der Krankh. der peripher. Nerven* (Wien, 1879) in the preparation of the account of Local Palsies.

CHAPTER II.

MOTOR EXCITEMENTS.

Disturbances of motility which are accompanied by an excess of motion may be well divided for clinical study into Convulsions, Spasms, Choreic Movements, Tremors, and Automatic Movements, to which I shall add, for convenience of description, Contractures, although the latter condition of the muscle might be very properly considered as essentially diverse from the other motor disturbances.

Convulsions.—A convulsion is a condition in which, owing to an excessive discharge of motor impulses from the nerve-centres, there is disturbance of the nervous system and usually a wide-spread excessive muscular contraction,—either a succession of violent momentary contractions and relaxations or a maintained contraction. When there is an alternation of contraction and relaxation, the convulsion or spasm is said to be *clonic*. When the contraction is maintained for a time, the convulsion or spasm is *tonic*. Convulsions are further characterized by being temporary states. They are naturally divided into general and partial.

In order to bring a case into the category of general convulsions it is not necessary that the whole of the muscular system should be involved, but only a sufficient proportion of it to make a wide-spread general disturbance. A partial convulsion is one which involves but a limited portion of the muscular system.

Spasms.—A spasm is a muscular contraction involving only a narrow territory, and not connected with a general involvement of the nerve-centres. The division between convulsion and spasm is to some extent arbitrary. In many cases the nature of the spasm is at once apparent. In the so-called Jacksonian epilepsy the convulsive disorder may for a time seem to be a local spasm, but its more serious nature is sooner or later shown by its connection with loss of consciousness. A spasm is a local phenomenon; and in any case, so long as there is no disturbance of other nerve-functions, a localized muscular contraction must be looked

upon as a spasm. It must, however, not be forgotten that an apparently simple local spasm may be the outcome of an hysteria, and be therefore the momentary expression of a general neurosis.

Choreic Movement.—A choreic movement is one in which irregular and more or less violent contractions occur, either in single muscles or in muscles which are associated in groups, so that a certain amount of resemblance exists between the diseased movement and the voluntary motion.

Tremor.—A tremor is a to-and-fro vibratile movement which is produced by more or less rhythmical successive contractions of antagonistic muscles. It does not in any way simulate voluntary movements.

Automatic Movements.—The term automatic movements is used to signify those motions which occur independently of the will of the person, but in which some voluntary act is closely simulated. An automatic act often involves an elaborate series of movements, such as occur in bowing, getting out of a chair, and the like. Cases of this kind belong in the class of *chorea major* of some German writers, but, as there is no relation between these motions and true choreic movements, I have preferred the term automatic movements.

CONVULSIONS.

Convulsions are divided into *epileptiform*, *hysteroidal*, and *tetanic*. In the epileptiform and hysteroidal convulsions there is a disturbance of consciousness. In the tetanic convulsion the nervous discharge comes solely from the spinal cord, and consciousness is undisturbed. In the epileptiform convulsion the disturbance of consciousness amounts to a complete suspension of it; whilst in the hysteroidal convulsion there is a peculiar condition in which consciousness is seemingly lost, although after recovery the patient remembers all that has happened during the convulsion, or in which the patient during the convulsion appears to be conscious, but after recovery has no remembrance of occurrences during the fit.

Epileptiform Convulsion.—In the typical, fully-developed epileptiform convulsion, the first symptom is a peculiar sensation first felt in some part of the body, and rising from its seat of origin up to the head, to be lost in unconsciousness. This so-called aura is

succeeded at once by the peculiar scream known as the epileptic cry,—a wild, harsh cry, probably due to a forcing of air, by convulsive contractions of the thoracic and abdominal muscles, through the glottis, narrowed by a rigid spasm of the vocal cords. With the first unconsciousness a general tonic spasm comes on, producing rigidity of the whole body, and violent distortions of the head, limbs, and face. The muscles of the trunk and abdomen are rigidly contracted. Often a turning of the head and eyes to one side is the first evidence of this condition, and in some cases not only the head but the whole body rotates. The facial muscles are violently contracted, usually most markedly on the side towards which the head turns; the jaws are fixed, and often drawn to one side; the arms are almost always flexed at the elbow, and still more strongly at the wrists, whilst the fingers are flexed at the metacarpo-phalangeal joints and extended at the others, the thumb being adducted into the palm or pressed against the first finger. The position of the fingers is similar to that of grasping a pen, and is due to conjoint spasmodic contractions of the interosseous and flexor muscles, as in the so-called athetosis. The legs are extended and the feet inverted. The position of the arms, legs, hands, and feet is usually that which is assumed in a case of universal tonic spasm, the members being drawn always in the direction of the muscles of superior power; but in some epileptic convulsions this is departed from, showing that certain of the muscles are more affected than others. Thus, the fists may be clinched, or the legs may be violently flexed and drawn up on the abdomen.

The stage of tonic spasm is usually accompanied by marked pallor of the face, and lasts from a few seconds to one or even two minutes, when it is succeeded by the stage of clonic spasm. Usually the coming on of this is marked by vibratory tremors passing into vibrations, which continually grow both slower and more severe until the intermissions become long and complete, and the limbs are alternately relaxed and jerked in movements as wild and bizarre as they are violent. During the period of clonic spasm the face becomes red, congested, even bloated, and often livid. The expression changes continually, since the spasm involves all the muscles of the face, including those of mastication and of the tongue, the soft palate, and the larynx. Owing to the violent working of the muscles of masti-

cation, the saliva is forced from the mouth in the form of froth. The tongue is continually thrust in and out by the spasm of its muscles, and is apt to be caught between the convulsively moving jaws and severely bitten. If the tongue happens to be between the teeth during the period of tonic spasm in an epileptic convulsion, it is bitten in the first stage of the fit.

The blood-stain which is so characteristic upon the froth is due to hemorrhage from the tongue. The pupils at the beginning of the fit are sometimes contracted; absolutely immovable dilatation occurs, however, very early, if indeed it be not present from the onset, and is the characteristic condition during the whole fit. The return of the pupils to the normal state is often one of the earliest evidences that the paroxysm has exhausted itself. In some cases after the fit the pupils undergo remarkable oscillations. During the height of the attack both the pupillary and the conjunctival reflexes are abolished. The sphincters are in the majority of epileptic convulsions not relaxed, but it is not rare for the urine and fæces to be passed, and Gowers affirms that this is more apt to occur in nocturnal fits. The pulse, feeble or unaffected in the beginning, during the height of the paroxysm is greatly increased in frequency and in force.

The stage of clonic convulsion lasts from three to four minutes, when it merges into the condition of quiet coma, and this in turn passes into a heavy sleep, which may continue for a few moments or for hours. After the waking the patient suffers from headache and general muscular soreness.

The description which has just been given represents the epileptiform convulsion as it is seen in what may be considered typical epilepsy; but even in the majority of cases of epilepsy some of the phenomena are wanting, and almost any of them may be absent. The essential or central idea of the epileptiform convulsion is the occurrence of complete unconsciousness, with nervous discharge taking the form of a clonic spasm, in which the movements have no relation, apparent or real, to those of ordinary life. The term epileptiform is used to represent any variety of such convulsions, because such convulsions occur most frequently in epilepsy; and, in a similar manner, the term hysteroidal convulsion is used to express a convulsion of the general character seen in hysteria. The distinction is, however, a somewhat arbi-

trary one, since every gradation between the two forms exists, and an hysteroidal convulsion may occur in true epilepsy and an epileptiform convulsion in hysteria.

Hysteroidal Convulsion.—In the hysteroidal convulsion the tendency is to a prolonged tonic contraction of the muscles, giving rise to the assumption of positions which bear more or less resemblance to those that may be taken in health. In the typical hysteroidal convulsion consciousness is impaired, but is not entirely set aside. Thus, a patient apparently unconscious during the fit narrates after recovery all that has occurred during the paroxysm; or, in other cases, what is known as *automatic consciousness* is present, in which the patient during the paroxysm seems to understand all that is said, but nevertheless after the paroxysm has no remembrance of what has occurred. The hysteroidal convulsion of the most highly developed and most pronounced type is usually preceded by some warning,—by a special feeling of malaise, epigastric sensation, palpitation of the heart, giddiness, constriction in the throat (the so-called *globus hystericus*), or, frequently, by an aura which appears to arise from the ovary, which under such circumstances is almost always hyperæsthetic. The patient falls, but usually gently and not with the suddenness of true epilepsy. Not rarely there is at this time an initial scream, which may be repeated during the paroxysms. The pallor of the face may now be marked. A simple tonic spasm develops, lasting two or three minutes: in it the limbs are usually rigid, with the toes pointed downward, and the arms extended or lying at the side of the patient. It is at this time that the respiration becomes arrested and there is developed the stage of asphyxia of some writers. The face is swollen, with turgid veins, and suffocation seems imminent. This condition may pass into the characteristic stage of opisthotonos, or may be followed by a furious clonic convulsion, in which bloody foam gathers about the mouth, although the movements preserve, to some extent, the appearance of wilfulness, so that the head or the arms are struck violently against the floor or dashed against pieces of furniture. Following these clonic convulsions, or not rarely replacing them, is the characteristic stage of opisthotonos, in which the person lying upon the back is bent violently into the arc of a circle, so that the body rests upon the head and feet, with the central portion arched from

the ground. The muscular contractions may be so severe that the head is drawn completely backward and the upper portions of the body rest upon the face, which looks towards the floor, whilst the lower end of the arc is supported on the toes. This condition of opisthotonos may last for some minutes. In some cases it is interrupted or replaced by violent, purposive clonic spasms, the patient suddenly leaping from the bed, or rising into a sitting position, and as quickly falling back again in opisthotonos. This to-and-fro movement may take place with extraordinary velocity. In some cases the body is bent violently laterally instead of backward. The opisthotonic stage may be interrupted by various emotional actions, or it may gradually subside into what may be called the emotional stage, when the patient assumes some attitude of intense emotion, and not rarely the so-called posture of the crucifix. In the latter attitude the subject lies upon the back, absolutely quiet, with the legs stretched out side by side and the arms firmly extended at right angles to the body, in the position of the cross. The widely-opened eyes, with dilated pupils, appear to be looking into indefinite distance, whilst a beatific smile is settled upon the face: so that by the ignorant the convulsant is often believed to be seeing visions of heavenly joy. Usually the emotion changes from time to time; the light of religious beatitude upon the countenance deepens into an intense voluptuousness, attended, it may be, with gestures and words full of venereal desire; or terror becomes supreme, and is manifested with equal intensity; or, in a passion of penitence, the convulsant, with sobs, bitter cries, and broken words, begs for mercy. Again the scene shifts, and, now singing, now weeping, reproaching alternately herself and her care-takers, the woman passes on to a slowly-perfected consciousness.

Hallucinations occur during and after the fit, and are always correlated to the emotional state: thus, during the terror, the subject sees rats and other disgusting objects, which, according to Charcot, are usually upon the side that is anæsthetic between the paroxysms.

The hysteroidal convulsion does not necessarily comprise all the stages or phenomena which have been just narrated. Indeed, convulsions which approximate the description just given are exceedingly rare in the United States, and, according to the state-

ments of English authors, also in Great Britain. The writings of the school of Charcot indicate that they are frequent in France. The difference appears to be connected with race, hysteria being more mild in the cold-blooded Anglo-Saxon. Dr. John Guitéras informs me that at Key West, where the inhabitants are generally of pure Latin blood, hysteria conforms with the descriptions of the French writers.

Characteristics of Hysteroidal Convulsion.—The varieties of hysteroidal convulsion as they occur in hysteria in the United States will be discussed in detail later in the chapter. Suffice it for the present to state that, as contrasted with the characteristics of the epileptiform convulsion, those of the hysteroidal convulsion are the peculiar disturbances of consciousness; the presence of emotional disorders; and the tendency of the muscular contractions to simulate in an exaggerated form natural movements and to become tetanic. Persistently clonic spasms pertain especially to the epileptiform convulsion, whilst tetanic rigidity is indicative of the hysteroidal.

Tetanic Convulsion.—In tetanic convulsion motor discharge arises from the spinal cord alone, the brain not being involved; consequently there are no disturbances of consciousness. The convulsions may be clonic or tetanic: they are evidently produced by irritation of the peripheral sensory nerves,—touching of the skin, draughts of air over the face, loud noises, or other feeble surface-irritations producing at once violent outbreaks. According to the cause of the tetanic convulsion is the amount of the muscular tissue involved.

EPILEPTIFORM CONVULSIONS.

The epileptiform convulsion may be due to—
First. Idiopathic epilepsy.
Second. Peripheral irritations.
Third. Cardiac failure.
Fourth. Organic disease of the brain.
Fifth. The action of certain poisons.
Sixth. Uræmia.
Seventh. Hysteria.

Idiopathic Epilepsy.

It is necessary to precede the discussion of the diagnosis between the etiological varieties of epileptiform convulsions by a study in detail of the convulsions as they occur in idiopathic epilepsy.

Epilepsy.—As seen in this country, the aura is wanting in a very large proportion of cases of true epilepsy. In the majority of cases, when present, it is connected either with one extremity or with the stomach, although psychical and special-sense auras do occur, and in some cases warnings are given by bilateral tremors or starts in the limbs, or by wide-spread indefinable sensations, which may perhaps be looked upon as generalized auras. Various as the auras are in different individuals, they are remarkably constant in the one sufferer, each epileptic paroxysm conforming to those that have preceded it.

An aura which commences in an extremity is usually first felt in the hand, but it may begin in the foot. From the hand it rises up the arm as an indescribable sensation, and is not rarely traced by the patient to the neck, where it disappears in the development of unconsciousness. The gastric aura is very frequent. It is variously described as pain, as burning or as a sense of coldness, as trembling, but more often as an indefinite distress. Usually there is no sensation of rising connected with it, but in some cases this occurs. An aura may be first felt in the chest, and ascend to the throat, when it gives rise to choking sensations. It may also begin in the face, tongue, larynx, pharynx, or indeed in any part of the body.

In psychical aura the emotion is almost always that of alarm or excessive terror. In very rare cases a very peculiar idea ushers in the epileptic convulsion, constituting a true intellectual aura.

Special-sense auras are rare, but do occur in connection with sight, hearing, smell, and taste. Of these special-sense auras the gustatory is the most infrequent, the ocular the most frequent. The ocular aura may consist in seeing colors; in an apparent increase or lessening in the size of objects; in indescribable visual sensations; in double vision, or in loss of distinctness of sight, deepening, it may be, into complete blindness. In a few cases there are actual visions, either simple or complex. When once a

certain personality, as that of an old woman, or of a man with hammer in hand striking a blow, has ushered in a paroxysm of epilepsy, the same form ushers in subsequent attacks. In the auditory aura, abnormal sounds are heard, such as hissing, or the whizz of rushing steam, or intermittent, pulsating noises, such as beating of drums or music, and, in very infrequent cases, even a spoken word. The olfactory aura seems always to take the form of a bad smell.

There are cases in which two auras coexist. Usually one of these is connected with the special senses.

Sometimes the warning preceding an epileptic paroxysm takes the form of a localized spasm, which may occur simultaneously with the sensory aura or without it. Usually, under such circumstances, the patient is suffering from organic brain-disease.

A very remarkable fact in connection with auras, especially those originating in the extremities, is that in many cases their upward passage can be arrested, and the fit aborted, by circularly compressing the limb above the aura. When the sensation reaches the point of compression it ceases, and the patient escapes. I have seen a similar occurrence in an epilepsy which commences with a motor contraction involving the three fingers. If this local spasm be immediately overcome by violently opening the clinched fingers, the further development of the paroxysm will be prevented. The arrest of the aura in this manner would seem to indicate that the starting-point of the epileptic paroxysm is in the periphery, where the aura is first felt; but even in some cases in which the epilepsy has been due to coarse lesion of the brain it has been found possible to prevent the paroxysm by checking the upward passage of the aura. To my mind, however, this does not absolutely prove, as seems to be believed by most recent authorities, that the fit does not really commence in the peripheral nerve-endings. For it is possible that in these rare cases of organic epilepsy the paroxysms are due to secondary changes which have been produced in the peripheral nerve-filaments. In the famous experiment of Dr. Brown-Séquard, section of the sciatic nerve in the guinea-pig produced structural alterations of the skin of the face and an epilepsy which evidently arose from the altered surface, and was cured by removing the diseased skin. With these experiments and facts it seems not at

all impossible that a disease of the brain may produce an alteration in the peripheral nerve-filaments of a distant part.

Convulsive Stages.—A brief tonic spasm may constitute the whole of the convulsive seizure. The clonic spasm of true epilepsy is especially characterized by its being universal, although one side of the body is often more strongly convulsed than the other. According to the elaborate studies of M. V. Magnon (*L'Épilepsie paralytique*, 1881), during the clonic stage of the epileptic convulsion the arterial tension and pulse-rate are greatly increased, but during the tonic convulsion the pulse-rate falls, and the rhythm is altered so that a complete systole and diastole may occupy six times the normal period. Afterwards the pulse gradually approaches the normal, or passes into a state of exaggerated force and frequency. During the clonic convulsion the respiration is noisy, stertorous, slow, or even irregular; often the pauses between the acts are so long that the patient seems to have stopped breathing, and when death occurs in a fit it is by the persistence of such arrest of respiration.

Petit Mal.—Almost any of the stages of the epileptiform convulsion may be absent in epilepsy. To the lighter paroxysm of the disease the name of petit mal has been given. In its more ordinary form petit mal consists of a momentary loss of consciousness, accompanied by pallor of the face, which is not, however, invariably present. The sufferer, in the midst of a conversation, suddenly stops, is quiet for a few seconds, and then takes up the thread of discourse as though nothing had happened, being in fact unconscious that anything has happened. Sometimes the period of consciousness is followed by a state of confusion of thought, and in other cases some peculiar sensation, or some more or less indistinct sensory or psychical aura, gives the patient warning of the attack.

The attacks range in degree of violence from the mildest petit mal to the most severe convulsions. Sometimes a slight unconsciousness is accompanied by a single loud, piercing scream, without further motor disturbance. Sometimes the epileptic paroxysm is comprised in a brief loss of consciousness, with irregular convulsive movements, or with just enough tonic contractions to cause the patient to fall. So variable is the epileptic attack in its manifestations that many authorities consider the

convulsion as secondary, and the unconsciousness as the essential portion of the paroxysm.

Not only the convulsive symptoms, however, but even the unconsciousness itself, may be absent in an epileptic attack. In a case which was probably one of epilepsy, and in which, so long as I had opportunity for watching the symptoms, there was no change, the patient had a distinct aura in the hand, rising up the arm in the usual manner, but suffering arrest in the neck, at which time, without any loss of consciousness, there were violent convulsive movements of the muscles below the position to which the aura had reached. Allied to this case are those described by Dr. S. Weir Mitchell in his work on Nervous Diseases, in which the whole paroxysm was sensory. In the most pronounced of Dr. Mitchell's cases an aura beginning at the feet passed up to the head, when it was lost in the sensation of a loud sound, like that of an explosion or of a pistol-shot, followed by a momentary sense of deadly fear. Dr. Mitchell states that in men he has never seen these paroxysms occur except in the border-land between waking and sleeping, but that in hysteric females the attacks may take place at any time. In some cases instead of the aura being lost in a loud noise it disappears in a flash of light, or in an excessively bad smell, or occasionally simply in a sense of a blow or of a shock on or in the head, or of a rending or bursting. It may be considered uncertain how far these cases ought to be ranked as epileptic. They seem to me to lie very close to epilepsy, and also to hysteria. Whatever hesitation there may be in classifying these cases, no doubt exists in regard to several cases that I have seen in boys, in which the paroxysms for a length of time consisted simply of a gastric aura, but finally developed into a complete epilepsy. Thus, a child eight years of age would cry out with a sudden painful sensation in his stomach, become excessively pallid, run to his mother and be held for a moment, when the whole attack would be over. In some of his spells he had tonic contractions of the feet and neck. According to the mother, they were not accompanied by loss of consciousness, but were followed by heavy sleep. Cases of this character show that we cannot consider loss of consciousness as essential to a paroxysm of idiopathic epilepsy.

As already stated, the especial character of the epileptic convul-

sion is the absence of apparent purposiveness in its movements: even this characteristic may be wanting; thus, in the frequently-quoted case reported by Professor Trousseau, a Parisian master-builder was habitually seized with attacks in which, although entirely unconscious, he would run from scaffold to scaffold, springing from plank to plank, but never falling. In a negro, long under my care, the epileptic paroxysm would begin with a scream; then the man would be seen running furiously; when seized and held, he would fall in a general convulsion. He himself stated that if he were permitted to have his run out, after going a quarter to half a mile, he would wake up without falling. Various maniacal outbreaks, or emotional disturbances, accompanied by automatic movements, may also replace an epileptic paroxysm. (See Disturbances of Intellection.)

For the reasons which have been assigned in the last few paragraphs, and which might be much more elaborated without adding to their force, it may be concluded that the essential character *of idiopathic epilepsy is a tendency to an abnormal discharge of nerve-force at irregular intervals, and without obvious cause, but dependent upon some persistent, almost irremediable, state of the nervous system.*

Peripheral Irritations.

A reflex convulsion is one which is due to some peripheral irritation. It is almost invariably epileptiform in its general symptoms, and may conform exactly to the typical epileptic attack. In the majority of cases, however, the convulsion is more prolonged and more purely clonic in its manifestations.

In some cases the supposed reflex convulsion takes upon itself the hysteroidal form; but under these circumstances it is exceedingly difficult to decide how far the convulsive seizures are really reflex, and how far they are due to a hysteroidal condition produced by the irritation. This is especially true of the numerous cases of the so-called reflex spasms, paralyses, and convulsions in children which are connected with irritation of the genital organs. In my own experience in young children, hysteria is more frequent in the male than in the female sex: it is also very generally connected with an early-acquired habit of masturbation. Even when no such habit exists, reflex disorders seem to be

produced by elongated prepuces, and are relieved by circumcision. I believe that not rarely the disease is of an hysterical nature, and is relieved in great part by the moral effect of the operation. I have seen also a general hysterical state accompanied by severe epileptiform and hysteroidal convulsions and distinct hysterical disorders of consciousness, imitation of animal movements, etc., produced in a boy by an injury to the head, and cured by removal of the cicatrix. Of course it is very difficult to say how far, under these circumstances, there was some irritation of the peripheral nerve-ending in the cicatrix; but there was no tenderness or other local manifestation, and all the symptoms partook of the hysteroidal type. We must conclude either that reflex epileptic attacks take on the hysteroidal form, or that hysteria may be produced by genital irritations in young children, and by the moral or physical effects of injuries.

There are, on the other hand, convulsions which conform to the epileptic type, and which are the result of an organic, peripheral irritation. Under these circumstances there is often, but not always, an aura arising at the point of irritation. In some cases there is tenderness at this point, or pressure upon it may produce an aura followed by an epileptic attack. The effect of ovarian pressure in some hysterical subjects shows how closely even this true reflex epilepsy is related to hysteria. In this so-called reflex epilepsy the irritating lesion may be in the extremities, or in the trunk, but in the majority of cases it is in the head. When the latter is the case, it is always doubtful how far the convulsive attacks are reflex, and how far they are produced by direct pressure or irritation of the nerve-centres. There is nothing in the reflex epileptic attack which points out its true character, except when there is a localized aura, or when the fit may be caused at any time by pressure upon the diseased periphery. The diagnosis is usually to be made out by the history of the case and the discovery of the point of irritation. In every case of apparently causeless repeated convulsive attacks, it is the duty of the practitioner to search carefully for any possible point of irritation, and if any depression of the skull be found, the operation of trephining should be performed, unless it can be clearly ascertained that the convulsive attacks preceded the injury of the skull. In children, especially in boys, it is imperative to examine carefully the genital organs.

Convulsions of Childhood.—A variety of reflex convulsions are the ordinary convulsions of children due to the irritation caused by teething, or by indigestible substances in the gastro-intestinal tract. In all cases of convulsive seizures occurring in young children in which there is no reason to suspect hemorrhage into the brain, epileptic disease, or an acute fever, and in which there is no point of irritation in the gums or elsewhere upon the surface of the body, an emetic should be at once administered. The symptoms of gastric convulsion are not peculiar, but usually there is no difficulty in arriving at a probable diagnosis if the following points are attended to:

First. The convulsion is not one of a series, and is general, and not accompanied by paralysis.

Second. It occurs in young children.

Third. There is no history of exposure to the contagion of scarlet fever or other similar disease, and no fever, excessive vital depression, or other symptom of a developing malignant fever.

Fourth. A history of over-eating, or of the use of indigestible food, such as stale cream-puffs, green fruit, candy, etc., may be attainable.

Relations between the Convulsions of Childhood and Epilepsy.—The question of the relations between epilepsy and the convulsions of childhood is one of great importance. As already stated, I do not believe that the diagnosis between these two affections is to be made out by the symptoms of a single fit. Moreover, it seems to me positively established by clinical experience that the tendency to convulsions in the child is closely associated with the epileptic diathesis, and that in many cases accidental convulsions are the commencement of a life-long epilepsy. In a very large proportion of the cases of epilepsy there is a history of repeated convulsions during early childhood, and there must be inherent differences in the nervous constitutions of children living under exactly similar conditions, some of whom frequently suffer from convulsions, whilst others pass unscathed. Some children are evidently born with the convulsive tendency, which in many cases is so firmly fixed in the nervous system that it cannot be affected by any mode of life or treatment: its possessor is doomed from birth to a hopeless epilepsy. I believe that there is a second class of cases in which the epileptic tendency exists, but in so

slight a degree as to be controllable by hygienic and medicinal treatment. Under these circumstances the child may suffer from repeated attacks of accidental convulsions and become epileptic, or by great care the early convulsions may be prevented, and the nervous system allowed to harden into the normal mould.

Pleuritic Epilepsies.—Among the class of reflex convulsions must at present be placed the affection sometimes known as pleuritic epilepsy, or, in some of its forms, as *pleuritic hemiplegia.* Cases of this condition were first reported by M. Maurice Raynaud in Paris in 1875, since which time a number of records have appeared in medical literature. The attacks have been caused by the injection of various substances into the pleural cavity for the relief of chronic pleuritic affections. They have followed the use of very weak solutions of iodine, chloral, carbolic acid, etc., and have not been due to absorption of the medicament. After the injection, suddenly the face becomes very pale, the respiration is suspended, and the pulse is very small and scarcely felt. Generally the spasms are first confined to the face or arm of the side in which the injection has been practised, but soon they become general, and are accompanied by profound unconsciousness. At first tonic, they almost always in a very short time become clonic. The pupils in the beginning of the attack contract, but afterwards dilate largely. The pallor of the face gives way to cyanosis as the respiration re-establishes itself and becomes stertorous. The urine and fæces are often involuntarily discharged, and the patient passes into a condition of epileptic coma, which may go off in half an hour, or may continue many hours, and in some cases has ended in death. A fatal result is usually preceded by a true epileptic status, with repeated clonic convulsions and even opisthotonos. A partial hemiplegia (see *Union Méd.*, 1876) has followed pleuritic injections without the occurrence of convulsions: less rare is it for a partial hemiplegia to be present after recovery of the patient from the epileptic paroxysm. The side in which the injection is practised is the one commonly affected. All the recorded cases of pleuritic hemiplegia have finally recovered. In the fatal cases of pleuritic epilepsy no lesions of the brain have been found.

Cardiac Failure.

Cardiac Epilepsy.—There is a class of cases in which epileptiform convulsions occur connected with marked disturbances of the circulation. The most prominent symptoms are attacks of unconsciousness, which are spoken of by some observers as apoplectic, and by others as epileptic, and a remarkable slowing of the pulse.

The habitual rate of the pulse has in the reported cases varied from twenty a minute up to fifty; usually it is between twenty-five and thirty-five. At the moment of the attack of unconsciousness the pulse-rate diminishes to ten, twelve, or even five per minute; and in the case reported by Dr. P. Thornton (*Trans. Clin. Soc.*, London, vol. viii. p. 95) it was proved by stethoscopic examination that in the first stage of the attack the heart ceased to beat for many seconds. In other cases the stethoscope has shown that the heart is acting feebly, although the pulse has been lost at the wrist. The respiration is at first usually quickened, and may become labored and stertorous. The face is very pale, but afterwards becomes congested and livid. The temperature is probably in most of these cases below the normal point, and, at least in some instances, falls very decidedly during the attack. It is quite common for the patient, directly before the epileptiform attack, to complain bitterly of intense coldness; and any case of alleged epilepsy in which the body during the attack is excessively cold is probably not idiopathic, but cardiac epilepsy.

Usually, during the stage of unconsciousness the patient lies quiet, but there may be very distinct general convulsions, or, more frequently, partial convulsions; and biting of the tongue was noted even by the earlier observers. (See W. Stokes, *Diseases of the Heart and Arteries*, p. 316.) In some of the reported cases the paroxysms have been ushered in by a distinct aura. On account of the conjoint occurrence of unconsciousness and convulsive movements, as well as of their irregular, persistent reappearance, these attacks are epileptic rather than apoplectic. In most instances the patient finally dies in a paroxysm.

It has been shown by the autopsies reported by W. Stokes and by Dr. A. R. Blondeau (*Études cliniques sur le Pouls lent permanent*, Paris, 1879), and by other physicians, that cardiac epilepsy is con-

nected with fatty degeneration of the heart. The close relation of the paroxysms to syncope is further evinced by the fact that in one of Dr. Stokes's cases the attack could be aborted by the patient's getting upon his hands and knees, with his head downward. Further, in a considerable number of the cases valvular murmurs have been heard. Charcot states that he has seen slow pulse in several old people in whom the heart, after death, was found to be normal; and cases were reported by Dr. Halberton (*Med.-Chir. Trans.*, vol. xxiv., 1841) in which the affection followed an injury of the neck, apparently as the result of a lesion in the medulla oblongata. It is possible that in some cases cardiac epilepsy is due to irritative lesions of the medulla.

Organic Disease of the Brain.

Almost any form of chronic or organic brain-disease may produce epileptic attacks, but clinical experience shows that the paroxysms are much more apt to be severe and pronounced when the upper brain is affected, and especially when the disease is situated in or near the motor region of the brain-cortex. Every variety of attack which occurs in idiopathic epilepsy is simulated in organic brain-disease. From the simplest vertigo up through petit mal to the most typical and violent epileptiform convulsions an unbroken series can be traced. Usually, however, certain features in the organic epileptiform attacks indicate the true significance of the convulsion.

At least in my experience, an aura connected with the special senses is in the majority of cases associated with some organic disease of the centres connected with such sense. When the organic brain-disease affects exclusively the motor region the convulsive paroxysm is not usually ushered in by a distinct aura, but in many cases both the sensory and the motor regions of the brain are implicated, and under these circumstances the spasm which begins the paroxysm is frequently associated with feelings of numbness or other paræsthesia in the affected part. Moreover, in some cases an aura occurs at a distant part of the body, and a true aura of the special senses, or even a psychical aura, may usher in an epileptic attack due to organic brain-disease.

The characteristic phenomenon of the *Jacksonian*, or organic, epilepsy is a continually-recurring tonic or clonic spasm of a

group of muscles. Almost any group of muscles may be affected, but the various paroxysms in the same case always begin in the same way. In studying an individual case it is necessary to observe especially the starting-point, the march, and the range of the spasm. There are three points at which it is not rare for a spasm to originate. The most frequent is probably the hand, then the foot, then the face and tongue. The range or spread of the convulsion varies from the slightest spasm confined to the thumb and index finger, and not accompanied with loss of consciousness, up to the violent general epileptiform convulsion.

According to their range, attacks are well divided into monospasms,—*i.e.*, spasm of the arm, face, or leg,—hemispasms, corresponding in range to hemiplegia,—and general convulsions. In the hemispasms the convulsions may be confined to the face, arm, and leg, but often there is in addition a turning of the eyes and head to the convulsed side, with more or less contraction of the respiratory muscles of both sides. This tendency of the respiratory muscles of the two sides to undergo consentaneous spasm is evidently etiologically connected with the fact that in hemiplegia they usually escape paralysis. In accord with the theory of Broadbent, which was explained in the chapter on Paralysis, one brain-hemisphere is evidently able to affect the respiratory muscles of each side.

Usually, if a spasm becomes general, after having affected the hand, it is by marching up the arm and down the leg, or up the leg and down the arm, and then crossing to the other side. There are cases in which the spasm begins not in the hand or foot, but in the shoulder or thigh, when the march of the spasm is down the limb. The relation of the spasms in these cases to the seat of the lesion is similar to the relation of paralysis to organic disease of the brain. This relation has been fully discussed in the chapter on Paralysis, and I shall not occupy more space with it: contenting myself with the statement that an irritative lesion in the motor region of the brain-cortex will produce spasms of those muscles which would be paralyzed by a totally destructive lesion of the same brain-centres. A lesion may partially destroy the functional power of a centre and thereby cause a partial local palsy, and at the same time irritate the remaining nerve-

cells and fibres, so that a Jacksonian epilepsy not rarely coexists with partial paralysis.

In Jacksonian epilepsy consciousness may or may not be lost. Usually, although not invariably, the degree in which consciousness is affected is in direct proportion to the severity and range of the convulsions: it will be often noted that, whilst consciousness is preserved in the early portions of the paroxysm, it is finally lost. When the portion of the brain affected is situated near the speech-centres, a temporary partial aphasia not rarely follows the fit. Indeed, as shown in the case reported by Dr. Allen Sturge (*Trans. Internat. Med. Cong.*, London, 1881), a sudden complete aphasia may form the most marked phenomenon in the organic epileptic attack, and may develop with the first convulsive movements, or, possibly, even precede them. More common still is a partial loss of power in the convulsed extremity, continuing for some hours after the passage of the paroxysm. When the convulsive seizure is narrow in its range, the weakness is apt to amount to a partial paralysis in the restricted region of the convulsion. When, however, the epileptiform seizure has been a very wide-spread and severe one, a hemiplegic weakness not amounting to a palsy is often noticed.

Severe general epileptiform convulsions often usher in an acute inflammation of the brain. Under these circumstances the delirium, the headache, the intolerance of light and sound, the fever, and the tendency to coma indicate the existence of a meningitis. Convulsions may also be a part of an attack of sunstroke. They are frequently present in tubercular meningitis. Whenever they are produced by a generalized disease of the brain they do not take on the Jacksonian type,—*i.e.*, they do not affect especially any group or associated groups of muscles, but are themselves generalized. Their causation is to be made out by paying attention to the symptoms associated with them.

Diagnosis between Idiopathic and Organic Epilepsy.—The diagnosis of an organic epilepsy can be usually made out with a fair degree of certainty by a study of the convulsive seizures. Before giving a positive opinion, however, it is usually wisest to wait for other manifestations of organic brain-disease. In idiopathic epilepsy the convulsive movements very rarely begin habitually in one extremity, and whenever convulsions have such

origin they are probably due to organic focal brain-disease. This probability approaches a certainty if the convulsive movements be entirely confined to one limb, one side of the face, or any other narrow muscular territory.

The age at which the epileptic paroxysm has first appeared is a matter of vital importance in the diagnosis between idiopathic and organic epilepsy. The statements of Gowers and of Hasse, including between them about two thousand five hundred cases of supposed idiopathic epilepsy, show that in seventy-five per cent. the disease commenced under twenty years of age. In Gowers's fifteen hundred cases, only about two per cent. began after the fortieth year, and about five per cent. after the thirty-fifth year. I believe that these small percentages would suffer still further reduction if there could be a rigid exclusion of organic cases; and it may be laid down as a rule of sufficient accuracy for practical diagnosis that *an epilepsy which develops after the thirty-fifth year of age is not idiopathic, but is due to some organic brain-disease, to the abuse of alcohol, reflex irritation, or other causes, which in some cases may be so hidden as to be exceedingly difficult of recognition.* An epilepsy which first appears after the thirtieth year should be viewed with great suspicion. In my own experience, epilepsy occurring after the age of thirty-five, not dependent upon assignable causes unconnected with organic brain-disease, has in at least eighty per cent. of the cases been due to syphilitic lesions of the brain.

Convulsions from Cerebral Hemorrhage.—An epileptiform convulsion may be produced by a cerebral hemorrhage. Under these circumstances the convulsive movements very frequently take on the form of a Jacksonian epilepsy, and may be confined to a group of muscles, to one extremity, or, more commonly, to one half of the body. The true nature of such a case can usually be made out without difficulty by attention to the evidences of local palsy. Thus, the face is drawn to one side, or an inequality of the pupils or of the movements of the eye can be made out, or, still more frequently, a distinct hemiplegia can be discovered, even though the patient be comatose. (See Apoplexy.) The apoplectic attack also usually occurs in advanced adult life, and is either the first convulsion the patient has had, or has been preceded by previous attacks of apoplexy. A cerebral hemorrhage may

happen during an epileptic fit. Under these circumstances the diagnosis can scarcely be made out until the recovery of consciousness, when the persistent paralysis will reveal the lesion.

Epileptiform Convulsions in General Paralysis.—Epileptic convulsions are a very common symptom of the disease known as general paralysis of the insane. They usually occur in the second or maniacal stage of the disease, and are still more frequent in the final stages of dementia. Many years ago Esquirol called attention to the fact that a succession of epileptic fits very frequently closes the scene in general paralysis. Epileptic convulsions may occur in the beginning of a general palsy, and may, indeed, usher in the disease. Under such circumstances their significance may readily be overlooked. This is especially the case when the major attacks are replaced by or associated with petit mal, in which the only symptom of the seizure may be a sudden pallor with mental confusion or a momentary unconsciousness, or a dilatation of the pupils with drawing of the head, or a sudden fixation of the countenance with an outpouring of cold perspiration, or an automatic repetition of coherent or incoherent phrases. Such paroxysms are apt to be interpreted as syncopal. Not rarely epilepsy in general paralysis takes upon itself the Jacksonian form, the convulsion being limited to isolated groups of muscles, or to one side of the face, one leg, or one arm, or being hemiplegic. Usually the attack begins with an aura, which is especially apt to be vertiginous. Sometimes the convulsion is preceded for several days by excessive restlessness, tinnitus aurium, and great psychical excitation. In other cases it begins with vomiting.

Very frequently there is a succession of convulsive attacks, producing a true epileptic status. In this condition the successive convulsions may be very different from one another, one being complete, the next partial, in one the head being drawn to the right, in the next to the left, and so on. Frequently after the paroxysms convulsive tremblings persist in the single muscles, or in groups of muscles, for many hours, and are followed by a more or less pronounced partial palsy. To use the words of Dr. Nicholl, paralysis follows the convulsion or spasm as the shadow follows the body. During the more severe paroxysms consciousness is always lost, but, especially when the convulsive movements

are more or less local, it may be perfectly maintained; occasionally it is affected as in hysteria. After severe seizures the mental condition of the patient is almost always distinctly aggravated.

It is affirmed by many authors that in general paralysis the temperature rises during and immediately after the epileptic paroxysms, and sometimes also immediately before them, and that the elevation continues for some hours, and may be very considerable after severe fits. This, however, does not seem to be universally the case, since Mendel (*Paralysis der Irren*, Berlin, 1880) affirms that he has repeatedly studied the temperature in cases in which there was a prolonged convulsive attack, and in which no elevation of temperature occurred. In a single case Mendel found that the temperature fell very decidedly as the result of an epileptic status which had lasted three hours. He also states that Westphal has noticed the same thing. Usually, however, a distinct sinking of the temperature in the course of an attack of unconsciousness during general paralysis marks the development of a true apoplexy.

Toxæmia.

Epileptiform convulsions may be produced by a very large number of poisons. The nature of such attacks is to be made out by a history of the case, and by the presence of other symptoms of the poisoning.

Convulsions in Fever. — Among the toxæmic convulsions are to be placed those which usher in scarlet fever and other malignant diseases. The diagnosis in these cases rests upon the recognition of the febrile disease. Excessive vital depression, with lowered or elevated bodily temperature; the absence of evidences of the convulsion being reflex, due to acute or chronic brain-disease, or uræmic; the age of the patient, and the history of exposure to the cause of an acute febrile disorder, are the principal facts which should lead to a correct diagnosis.

Alcoholic Epilepsy.—There are two distinct epileptic conditions produced by intoxicating drinks. In one of these the convulsions are symptomatic of acute poisoning, and come on during an orgy, or immediately after a single excessive draught of liquor.

In the second form the convulsions are apparently not the im-

mediate result of alcohol in the blood, but are developed at a time when the system is not profoundly under the direct influence of the poison. These epileptic convulsions may supervene during delirium tremens, when they are accompanied by hallucinations; during the mental enfeeblement of profound chronic alcoholism, when they are associated with dementia, paralysis, or stupor; or at a time when the general symptoms of chronic alcoholism are not pronounced. In the alcoholic convulsion the symptoms may closely resemble those of true epilepsy, and not rarely the attack is ushered in by headache, gastric embarrassment, troubles of vision, excessive tremors, or some similar prodrome which may be looked upon as partaking of the nature of an aura. The convulsions usually occur in paroxysms,—two, three, four, or more, one after the other, at intervals of a few minutes.

Not only may major epilepsy be closely simulated by the alcoholic affection, but simple epileptic vertigo or true petit mal may exist, either alone or associated with the major convulsions.

Alcoholic epilepsy is often associated with hallucinations, especially of terror, and the convulsion is not rarely followed by temporary mental derangement, which may last only for a few minutes or may continue for hours or days. The mental derangement may take the form of an acute dementia, in which the intellectual functions seem to be in abeyance, and the subject is reduced to the condition of an automaton, obeying immediately and mechanically all commands and impulses from without. This state of perverted consciousness has, in some instances, lasted for days. Suicidal impulses are very frequent.

There is nothing diagnostic in the convulsion of alcoholic epilepsy, and it is especially important to remember that if under the prolonged use of the stimulant the epileptic paroxysms have occurred repeatedly, they may continue even if the patient cease to use intoxicating liquors. Under these circumstances it must be considered that an idiopathic epilepsy has been produced by alcohol. In every case of apparently causeless epilepsy occurring in middle life, the possibility of its being alcoholic must be borne in mind.

Uræmia.

Uræmic Convulsions.—A very frequent cause of epileptic convulsions is uræmia. In this disease the convulsive phenom-

ena may be altogether wanting during the attacks of coma, or may appear only in the shape of twitchings of various groups of muscles; but severe convulsions of the epileptic type are frequent. In these there are usually complete insensibility, rotatory movements of the eyes and head, violent clonic contractions of all the muscles, biting of the tongue, foaming at the mouth, and finally coma: so that it is not rarely impossible from the convulsion alone to decide that the attack is not true epilepsy. Even when disease of the kidneys has not been previously suspected, a history of prolonged dyspepsia, frequent vomiting, occasional attacks of asthma, failure of general health, etc., may generally be obtained, and an examination of the urine will usually reveal the nature of the case; but sometimes such examination must be made repeatedly before evidences of kidney-disease can be found. The danger of overlooking the serious nature of uræmic eclampsia is especially great when the convulsive seizure takes on the hysteroidal type. So long ago as 1840, Dr. Bright described cases of uræmia in which there were furious convulsions without loss of consciousness; and Dr. Roberts has reported similar instances. I have seen the diagnosis of hysterical convulsions persisted in by practitioners of large experience until within a few hours of death. In all cases of convulsions occurring for the first time during adult life, a very careful study of the urine is essential.

Moreover, fatal uræmia may occur with a urine that is apparently normal, and in a large proportion of cases of contracted kidney albumen is absent from the urine for long periods. The study of the specific gravity of the urine is of the utmost importance, and in doubtful cases the percentage of urea should be determined. Unless diabetes insipidus exists, a specific gravity habitually below 1010 is almost proof of the existence of chronic Bright's disease. In doubtful cases a number of examinations of the urine taken at different periods of the day should be made. Fasting urine may be found to be of abnormally low specific gravity, and urine voided three or four hours after a heavy meal to contain albumen. The power of elimination of the iodide of potassium has been proposed as the means of testing the renal secreting surface. It is affirmed that, after a full dose, this drug can, in an hour, be readily recognized in the urine by adding nitric acid and then starch, but that when contracted kidney exists the iodide

fails to appear, or comes over in only very small quantities and after a very long interval.

HYSTEROIDAL CONVULSIONS.

The symptoms of hystero-epilepsy have been sufficiently detailed in the discussion on the hysteroidal convulsions. It was there shown that the attack is usually ushered in by an aura, and is made up of peculiar disturbances of consciousness, of emotional storms, and of clonic and tonic convulsions, simulating to a greater or less degree purposive movements.

The major hysterical convulsion varies almost indefinitely. The ordinary hysterical attack which we see habitually in the United States may be well spoken of as minor hysteria.

Minor Hysteria.—Into minor hysterical paroxysms enter all the elements of the major affection; but usually some of the symptoms are wanting in individual attacks, and not rarely a single stage constitutes the whole paroxysm.

The aura is not usually present, unless the so-called *globus hystericus* be considered to represent it. The globus is, however, a local convulsion, and is not necessarily prodromic. It is described as a feeling of a ball in the throat, or of something rising in the throat, and is the result of spasm of the muscles in and around the larynx. In ordinary minor hysteria the emotional state is usually well developed, and is especially prone to express itself by uncontrollable laughter or equally uncontrollable sobbing or crying. A very characteristic performance which I have seen, especially in young boys, is that which may be termed *beast-mimicry*, in which the patient bites or snaps or snarls like a dog, or crows like a cock, or in some other way imitates the movements and the vocal acts of the lower animals. Among these cases belong the not rare attacks of *spurious hydrophobia*, in which, either with or without severe general convulsion, the subject shows profound dread of water, great emotional disturbance, often crying out to be held lest he bite some person, and continually snarls and barks and attempts to bite. These symptoms do not closely resemble those of true hydrophobia, in which disease the subject never offers to bite, and does not make any noises resembling those of the dog or any other lower animal. Beast-mimicry may be considered as diagnostic of hysteria.

The convulsive symptoms of minor hysteria are tonic rather than clonic. More or less persistent rigidity is very frequent and very characteristic. It may last for hours, or may pass by in a few moments. The disturbances of consciousness take the form which has already been fully described in speaking of the major attacks. Occasionally the abolition may be complete, but almost invariably at some stage of the attack the characteristic alterations of consciousness can be discovered. The diagnosis between fully-formed hysteroidal epilepsy and idiopathic epilepsy is, of course, easy, but I have seen in private and dispensary practice patients suffering from recurrent irregular attacks whose nature remained very doubtful for months. One great difficulty in these cases is that the physician is usually unable to see the patient during the paroxysm. A further complication arises from the fact that severe hysterical manifestations may follow immediately upon an epileptic fit, and that hysterical convulsions may alternate with those of true epilepsy. The only safety is to be found in a very cautious diagnosis and prognosis, especially when the subject is a young woman. In such patients I have several times seen "spells" which I had believed to be epileptic yield to careful treatment.

Temperature as an Aid to Diagnosis.—Aid in the diagnosis between uræmic convulsions, hystero-epilepsy, and epilepsy can sometimes be obtained by a study of the temperature. In 1865, Dr. Kien (*Gaz. Méd. de Strasbourg*, 1865, p. 12) called attention to the fact that even when the uræmic convulsions are very violent they are accompanied by a marked fall of temperature, which increases until death. This has been confirmed by Roberts, of Manchester, by Hirtz, Hutchinson, Charcot, Teinurier, and Bourneville. (See *Études clin. et therm. sur les Maladies du Syst. nerveux*, Paris, 1873.) In the severe isolated attack of epilepsy there is usually a distinct elevation of temperature. In the severe isolated hysteroidal convulsion the temperature also rises, but not so distinctly as in true epilepsy. In the epileptic state—*i.e.*, in that condition in which there is a prolonged series of fits, connected by coma and occurring at short intervals—the temperature rises steadily throughout the whole condition, this rise of temperature not being preceded or interrupted by a fall. On the other hand, when the series of convulsions are the expression of a hystero-epilepsy, the temperature falls very rapidly imme-

diately after each convulsion, and does not in the successive attacks reach distinctly higher than in the first convulsion. In a supposed hystero-epilepsy a continuous elevation of temperature is sufficient to establish the presence of some other disorder, or at least of a very threatening condition of the nerve-centres. Thus, in a curious case reported by Quincke (*Arch. der Heilkunde*, 1864), after a series of apyretic hysteriform convulsions, convulsions occurred with an elevation of temperature to 43° C., and soon ended in death. Puerperal convulsions are usually supposed to be uræmic, but according to the researches of Bourneville they are distinctly separated from uræmia by the fact that the temperature rises almost brusquely in the beginning of the convulsions, and, when the latter are frequently repeated, maintains itself at an elevated position with great steadiness. Each paroxysm is marked by a slight increase of heat, and if the attack is to end fatally the temperature finally becomes very high; if recovery occur, the temperature gradually falls. I am, however, doubtful whether the temperature *always* falls in true uræmia. I have certainly seen it rise in coma occurring in persons suffering from contracted kidney, and apparently uræmic. The subject is one of much interest, and needs further clinical study. It is also complicated by the frequency of serous or true apoplexy in uræmia. Clinical studies of the temperature should, as far as possible, be checked by post-mortem results.

TETANIC CONVULSIONS.

The tetanic convulsion is one in which the cerebrum does not participate, and in which, therefore, consciousness is not disturbed except secondarily as the result of asphyxia.

It may be due to—

1. Hysteria.
2. Tetanus.
3. Poisoning.
4. Tetany.

Hysteria.—Hysteria may produce convulsions which conform to the tetanic type, but usually, sooner or later, the cerebrum is involved, and the characteristics of the hysterical convulsions appear. The diagnosis of hysterical tetanus is fully discussed on page 126.

Tetanus.—Tetanus is characterized by tonic contractions of many of the muscles, associated with violent paroxysms of reflex convulsions affecting the whole body. There are two distinct etiological varieties of it,—the traumatic and the idiopathic. The traumatic disease is due to a traumatism, usually a laceration, although it may follow a blow, or may occur after childbirth. Idiopathic tetanus in many instances comes on without obvious cause, but not rarely follows exposure. There are no distinctive symptoms separating the two varieties, except it be in that the idiopathic disease is less violent and less frequently fatal than is the traumatic affection. The diagnosis between the two varieties must rest upon the history.

Tetanus usually begins with mild symptoms, which gradually increase, although in rare cases the attack is sudden and the course of the disease very short: thus, in a patient of Groetzner, the convulsions set in the moment a ligature was tied around the crural nerve, and proved fatal in six hours. Jaccoud speaks of a case reported by Bardeleben, in which a negro died of tetanus fifteen minutes after receiving a wound. On the other hand, tetanus may not appear until long after the reception of the injury, and may run a very prolonged course, with active symptoms lasting for four or five weeks.

The first manifestation of the disorder is usually a stiffness of the muscles of the lower jaw, of the œsophagus, and, in a little while, of the neck. At first the jaw can still be opened, but in a short time the masseters set so firmly in tonic spasm that the upper and the lower teeth are immovably pressed together. At the same time the spasm of the œsophagus increases so that swallowing becomes very difficult and fatiguing. The speech is altered in distinctness and in tone, not only because the lips and the tongue are rigid, but also because the muscles of the larynx are implicated. The facial muscles of expression are very rapidly involved; the forehead is wrinkled; the eyebrows are drawn upward, with the eyeballs staring and motionless and the pupils generally contracted; the corners of the mouth are pulled outward and downward, and the lips parted so that the teeth are exposed, whilst the naso-labial folds are exaggerated. As the spasms persist, the face sets itself into an anxious, frightened, wildly-excited, half-laughing, half-crying expression, to which

the name *risus sardonicus* has been given. Spasm of the muscles of the neck soon succeeds, if it does not accompany, the first evidences of the disease, and in a very short time the whole erector spinæ group are involved. Somewhat later the muscles of the legs and of the arms are affected, the tonic spasms being more marked in the legs, and in some cases the forearms and the hands entirely escaping. Painful spasmodic erections of the penis may now occur.

When the tonic spasms are thoroughly established, the body assumes a characteristic position: the head is drawn back; the trunk arched into opisthotonos; the belly hard and spasmodically retracted; the legs rigidly contracted. Paroxysms of violent increase of the tonic spasms now occur, or brief, furious discharges of nerve-force give the appearance of rapidly-repeated clonic convulsions. During the paroxysm the distorted position of the body is greatly increased. With a sudden shuddering the opisthotonos becomes marked, the head is forced back into the pillow, the flattened abdomen is thrown forward, and the feet are driven into the bed, so that the body rises until it assumes the form of a bow resting upon the head and the feet. The spasm of the respiratory muscles and of the pharynx and glottis arrests respiration, and causes not only a sense of suffocation but an absolute cyanosis, which may continue until unconsciousness and even death result. Usually during the attack the thorax is spasmodically bound in the position of inspiration and the diaphragm is rigidly drawn downward. The respiration, arrested during the paroxysm, may be rapid between the convulsions; the pulse is sometimes not affected, but usually becomes more and more rapid, and the arterial pressure, at first high, especially during the convulsion, falls as exhaustion sets in. The skin is bathed in sweat, whilst from the bronchial mucous membrane there is usually an outpouring which, as coughing is impossible, may accumulate in the bronchial tubes and be a very distinct factor in the production of suffocation.

The temperature in some cases of tetanus does not rise distinctly, but very frequently it steadily mounts upward; and in fatal cases as death approaches this rise may suddenly become extreme, occasionally reaching, according to the observations of Wunderlich, 113° F. A remarkable but well-authenticated phenomenon is

that after death from tetanus the temperature of the body often continues to rise for a time.

The paroxysms of tetanus are reflex, and are provoked by the slightest peripheral irritation: a loud sound, a bright light, a draught of air, a new contact with the bedclothes, intestinal peristaltic movements, an irritation so slight as in health not to be noticed, may cause an immediate furious convulsive outbreak.

The sufferings during an attack of tetanus are extreme. During the whole course of the disease there is usually excessive sleeplessness, and the patient is tormented not only by pain, but also by distressing thirst, and in some cases by a very acute hunger, both the thirst and the hunger being, in a measure, due to the difficulty of opening the mouth and of swallowing. The cramp-pain is horrible, is more severe during the paroxysms, and is in direct proportion to the severity of the contractions. The effects of these sources of distress are increased by the characteristically clear mental state. In scarcely any other disease is there so much anxiety and dread. Retention of urine is frequent, but, especially in children, involuntary micturition may occur; rarely the urine is passed in large quantities, usually being distinctly below the average amount; it is of high specific gravity, and of a dark brown color. Sugar and albumen are exceptionally present.

Trismus Neonatorum.—A variety of tetanus very rare in this country is the so-called trismus neonatorum, which occurs in new-born babes. In it the spasms are confined chiefly to the muscles of the jaw. Some authorities believe that it is a traumatic tetanus due to irritation of nerve-filaments in the stump of the umbilical cord; others, that it is produced by pressure upon the medulla during labor; others, that it is due to phlebitis and other pyæmic manifestations. Like other forms or varieties of tetanus, it is connected with epidemic influences, and is notably worse in extremely hot or extremely cold weather. It is probable that the starting-point varies in different cases, and that the epidemic or atmospheric influences act as strong predisposing causes.

Differential Diagnosis of Tetanoid Affections.—The only diseases which could possibly be confounded with tetanus are tetany, meningitis, strychnia-poisoning, and hysterical convulsions. Cerebro-spinal meningitis is distinguished at once by the cerebral symptoms; whilst in a pure spinal meningitis the radiating pain

along the nerve-cords ought to reveal the nature of the affection. The diagnosis between tetanus and tetany is given on page 128.

About fifteen years ago, in a very important medico-legal case, there was pronounced disagreement between the medical experts in regard to the nature of certain convulsions. The following table, which I published shortly afterwards, was based upon the experiences of that trial. Time has confirmed its correctness. I have since seen one case in which a broker, after prolonged excitement on a very hot day, was suddenly seized with nausea, directly followed by general convulsions closely conformable to those detailed in the second column. These convulsions were undoubtedly hysteroidal, provoked by exhaustion and gastric irritation.

Tetanus.	Hysterical Tetanus.	Strychnia-Poisoning.
	Commenced with blindness and weakness.	Begins with exhilaration and restlessness, the special senses being usually much sharpened. Dimness of vision may in some cases be manifested later, after the development of other symptoms, but even then it is rare.
Muscular symptoms usually commence with pain and stiffness of the back of the neck, sometimes with slight muscular twitchings; come on gradually.	Muscular symptoms commenced with rigidity of the neck, which gradually "crept over the body," affecting the extremities last.	Muscular symptoms develop very rapidly, commencing in the extremities, or the convulsion, when the dose is large, seizes the whole body simultaneously.
Jaw one of the earliest parts affected; rigidly and persistently set.	Jaws rigidly set before a convulsion, and remained so between the paroxysms.	Jaw the last part of the body to be affected; its muscles relax first, and even when during a severe convulsion it is set, it drops as soon as the latter ceases.
Persistent muscular rigidity, very generally with a greater or less degree of permanent opisthotonos, emprosthotonos, pleurosthotonos, or orthotonos.	Persistent opisthotonos and intense rigidity between the convulsions, and after the convulsions had ceased the opisthotonos and intense rigidity lasted for hours.	Muscular relaxation (rarely a slight rigidity) between the convulsions, the patient being exhausted and sweating. If recovery occur, the convulsions gradually cease, leaving merely muscular soreness, and sometimes stiffness like that felt after violent exercise.
Consciousness preserved until near death, as in strychnia-poisoning.	Consciousness lost as the second convulsion came on, and lost with every other convulsion, the disturbance of consciousness and motility being simultaneous.	Consciousness always preserved during convulsions, except when the latter become so intense that death is imminent from suffocation, in which case sometimes the patient becomes insensible from asphyxia, which comes on during the latter part of a convulsion, and is almost a certain precursor of death.

Draughts, loud noises, etc., produce convulsions, as in strychnia-poisoning.	Desired to be fanned.	The "slightest breath of air" produces convulsion.
May complain bitterly of pain.	Crying-spells, in which he "sobbed violently," and "cried like a child," alternated with convulsions.	Patient may scream with pain, or may express great apprehensions, but "crying-spells" would appear to be impossible.
Eyes open, rigidly fixed during the convulsion.	Eyes closed.	Eyes stretched wide open.
	The spasm in leg must have been partial, as the feet were crossed and toes inverted, which could not happen if all the muscles were involved, because the muscles of eversion, being very much the stronger, would of necessity overcome the antagonistic muscles, and the feet be everted.	Legs stiffly extended, with feet everted, as the spasms affect all the muscles of the leg.

Tetany.—Under the name of Intermittent Tetanus, Dance originally described the convulsive disorder now usually known by the title given to it by Corvisart,—*i.e.*, tetany. It is a disease of childhood and early adult life, and is exceedingly rare in this country. If the various writers upon the subject are to be credited, tetany may be produced by excessive lactation, by the puerperal state, by exposure to cold, by prolonged fatigue, by exhaustion from diarrhœa or other cause, by the irritation of intestinal worms, by exposure, and even by the rheumatic diathesis or the infectious fevers. Further, it is affirmed that it may result from excessive emotion, and spread from patient to patient as an epidemic,—facts which would indicate an hysterical nature. It consists essentially of successive tetanic convulsive attacks, separated by intervals of quiet and repose. The paroxysms may continue for some minutes or for many hours, and may cease gradually or abruptly. Arthralgic pains, formications or numbness in the hands, radiating pains in the fingers, temporary partial blindness, headache, sense of fatigue, etc., are assigned as occasional prodromes. Usually the spasms are most marked in the upper extremities, and sometimes are confined to them; the fingers are drawn together so as to form a cone like the hand of the accoucheur when he is about to introduce it into the vagina. Rarely there is a more accentuated flexion of the fingers, and still more infrequently the hand and the fingers are stiffly extended.

The feet may be attacked; sometimes cramps of the calf occur

without distortion, but in other cases the feet are violently extended, with the toes pointing downward, or very rarely they are flexed. The thigh usually escapes, but spasm of the abductors and crossing of the feet have been noticed. It is affirmed that the spasms may be confined to the arms, but that the legs are never the only portion of the body attacked. Only in the severest cases are the trunk-muscles affected; but opisthotonos and menacing dyspnœa do occur. Even more exceptional than these are spasmodic closures of the jaw and distortions of the face. The course of the disease may be painless; sometimes, however, neuralgic pains run along the nerves, and usually cramp-pains are present in the affected muscle. Anæsthesia and analgesia are ordinary phenomena. According to Erb, the faradic excitability of all the muscles of the body is increased. The course of the disease is often slow, may continue for months, and usually ends in recovery. A diagnostic sign first discovered by Trousseau is that during the periods of relaxation, and in some cases even as long as three days after the occurrence of a convulsion, the latter can be brought on by pressure upon the principal nerve-trunks or blood-vessels. By this symptom, by the complete relaxation between the attacks, and by the partial character of the convulsion, tetany is at once distinguished from tetanus.

LOCAL SPASMS.

For the purposes of diagnostic study local spasms are usefully divided into those which affect the muscles of organic life and those which attack the voluntary muscles.

Spasms of the Muscles of Organic Life.

Œsophageal Spasm.—In spasm of the œsophagus the diagnosis of the nature of the stricture can usually be made out without trouble; but, if there be a reasonable doubt, the patient should be etherized, when, if the stricture be spasmodic, a probang can be passed into the stomach without the resistance which would be met with in organic disease. The spasmodic affection develops rapidly, and usually without a history of the causes which produce œsophageal lesion: it is mostly painless, varies excessively from time to time, and is apt to disappear suddenly. It is usually accom-

panied by distinct hysterical or neurotic symptoms, or occurs in a person who has such a history.

Rectal Spasm.—Rectal stricture is occasionally so closely counterfeited that the diagnosis can be made out only by physical exploration. Under these circumstances the flattening of the stools and the other evidences of organic disease are produced by a spasm of the sphincter ani. Spasm of the rectum and of its sphincter is very prone to be associated with vaginismus and spasmodic dysmenorrhœa. It also is occasionally produced by the pressure of a misplaced uterus upon the sacral nerves.

Urethral Spasm.—Urethral spasm is usually caused by some irritant substance in the urine, such as cantharides, but in some cases is hysterical in its origin. It is to be recognized by its sudden development, and by the absence of a history of gonorrhœa. Etherization will always decide a doubtful case.

Vomiting.—A symptom which may be considered as a form of local spasm is vomiting. Vomiting of course accompanies many acute fevers or disorders, but in its chronic form it is usually either due to disease of the stomach, liver, or kidneys, or of the brain, or is hysterical. In any case of chronic vomiting it is essential to determine at once the condition of the stomach, liver, and kidneys.

Uræmic vomiting is accompanied by dyspeptic symptoms, is very obstinate, and generally is associated with headache, asthma, or other uræmic manifestations. Its diagnosis rests chiefly, however, upon the detection of the disease of the kidney.

The cause of chronic gastric vomiting is sometimes difficult of determination. It is necessary to examine for the existence of a tumor, or of pain and tenderness, or of blood in the matters vomited. In ulcer of the stomach the pain is habitually referred to the back, between the shoulders, or sometimes as low down as the lumbar region. In hysterical vomiting there is frequently pain in these parts, and not rarely blood will be found in the vomit. The practitioner is especially liable to be deceived when along with these manifestations there are gastric tenderness, excessively foul tongue, and pronounced dyspeptic symptoms. The hysterical pain in the back is usually, if not always, associated with marked superficial tenderness, or hyperæsthesia, which is wanting in the organic disease. In cases of hysteria the diagnosis of gastric ulcer should be made with reluctance.

Tumors, inflammations, and other diseases of the brain may produce vomiting. There is nothing in the vomiting which is characteristic, unless it be its apparent causelessness, and the fact that in many cases it is affected by rapid change of posture: thus, vomiting, which is not present so long as a horizontal position is quietly maintained, may be at once provoked by the subject's rising suddenly from the bed. The diagnosis of nervous vomiting is in most cases to be reached chiefly by the process of exclusion, and by the discovery of symptoms, other than the vomiting, pointing to disease of the brain.

Nervous Cough.—A form of repeated rhythmical spasm which is especially seen in persons suffering from minor hysteria is the so-called nervous cough, which may exist independently of catarrhal or other organic irritation of the respiratory tract, or may be associated with such irritation.

The cough is usually ringing, dry, and very loud, often rasping, and apparently excessively annoying, and is prone to occur in violent paroxysms. It must not be forgotten that not rarely the catarrhal and nervous elements are associated. Under such circumstances the treatment, to be successful, must be directed to each element of the condition. The diagnosis of nervous cough is arrived at chiefly by exclusion,—*i.e.*, by finding that cough in a neurotic subject is entirely disproportionate to the amount of catarrhal disease.

Phantom Tumor.—A phenomenon which may be considered the result of a conjoint spasm of muscles of voluntary and involuntary life is the so-called phantom tumor. This occurs almost exclusively in neurotic women, and consists of an apparent local swelling and hardness in the abdomen, which on palpation gives to the hand a sensation like that imparted by a true abdominal tumor. Its true character is usually at once revealed by the absence of percussion dulness; but it is said that organic percussion dulness may be simulated. Abdominal section has been practised for the relief of a phantom tumor due simply to a local spasm of the abdominal walls, and probably also of the intestines. In all cases of alleged or apparent abdominal tumors in hysterical women suspicion should be aroused, and, if there be any doubt, the patient should be etherized, when the nature of the alleged growth will be revealed by its disappearance or its permanence.

Spasms of Voluntary Muscles.

Laryngismus Stridulus.—A very important form of local spasm is the so-called spasm of the glottis, or laryngismus stridulus, which is probably the result of contraction of the two thyro-arytenoid and the two lateral crico-arytenoid muscles and the arytenoideus muscle, and is due to an irritation of the recurrent laryngeal nerve. Laryngismus stridulus is a very rare affection in adults, and when it occurs in women is usually hysterical. In men it is of very serious import, as in most cases it depends upon dangerous organic disease. The attacks come on without warning, and may occur during either the night or the day. In the mildest form the child throws itself backward, grows pale, and makes uneasy movements with its extremities. In a few seconds the attack is over; the child is left irritable, and is sometimes punished for naughtiness. In the more violent paroxysms there is whistling or crowing inspiration, preceded by irregular, laborious, and audible expiration, or even by a momentary arrest of respiration. In the severest paroxysms the closure of the glottis is complete, so that the thorax, diaphragm, and anterior abdominal muscles remain immovable. The child, exceedingly pale, and with a wild expression of anxiety in its countenance, throws its head back, with the mouth wide open, and the nostrils dilated. The face now rapidly becomes cyanotic; a cold sweat covers the forehead, and the blue lips purse out in a manner almost pathognomonic. If the spasm continue, unconsciousness and convulsive symptoms rapidly develop.

The muscular contractions may take the form of tonic cramp of the lower legs with abduction of the big toe, or other distortions about the feet; sometimes the arms are similarly contorted. Not rarely general tonic and clonic convulsions, with involuntary discharges of urine and fæces, occur. This condition of unconsciousness and convulsions may last from a few seconds to two minutes, and occasionally ends in death. Usually crying or whistling inspirations mark the beginning of the relaxation of the spasm; the irregular inspirations become rapidly normal; the feeble, quick pulse regains its ordinary character, and the general cyanosis passes off. Very frequently after the paroxysm the child goes to sleep. If it does not, it is invariably peevish.

Laryngismus stridulus can hardly be confounded with any other disease. It is to be distinguished from catarrhal croup by the sudden beginning of the attack; the whistling or crowing inspiration; the noisy expiration; the increasing cyanosis; the frequent loss of consciousness, and general convulsive phenomena; the feverless course; the short duration of the paroxysms, and their termination through convulsive crowing inspiration; and especially by the absence of cough and other evidences of catarrhal inflammation of the larynx between the paroxysms. In the majority of cases the disease is connected with rachitis, and it is closely allied to rachitic epilepsy. In some cases the spasms seem to be due to reflex irritation from teething; or they may be the result of irritation of the nerves by enlarged glands; and especially in adults it is necessary that the larynx be explored for evidences of local ulcerations. In extremely rare cases the symptoms are said to have been dependent upon local disease of the medulla oblongata.

Occupation Neuroses.—Under the name of occupation neuroses may be associated diseases which are connected with the excessive use of localized groups of muscles in business or professional occupation. Because the symptoms are most frequently seen as the result of the excessive use of the pen, the disease is typified in the so-called writer's cramp; but it also occurs among telegraphers, dancers, pianists, workers in metal, etc., when it is known as telegrapher's cramp, dancer's palsy, hammer palsy, etc.

The symptoms are undoubtedly produced by the excessive repetition of movements requiring exceedingly fine co-ordination, and differ from the simple muscular exhaustion which occasionally is produced by severe, gross muscular efforts. In 1868, Moritz Benedict stated that there were three forms of occupation neuroses,—the paralytic, the spasmodic, and the tremulous. These varieties undoubtedly exist in nature, although not absolutely separated from one another,—the distinction between them being simply that in some cases the paralytic symptoms are most marked, whilst in others the spasm or the tremor is the most pronounced. According to my own observation, the paralytic form of the affection is much the most frequent, although some authorities assert that the spasmodic is the ordinary variety.

I shall take the *writer's cramp* as a type of the occupation neuroses. In the paralytic form of it the first symptom is usually a painful feeling of fatigue in the arm, which is often associated with formications and numbness, but usually not with true anæsthesia or hyperæsthesia. Only in rare cases can tenderness be found over the nerve-trunks. The pain is always increased by writing, and at last it grows so intolerable as altogether to forbid the use of the pen. With this fatigue and pain there is usually a sense of stiffness, and often a distinct muscular resistance when the effort is made to grasp the pen. At first no pain is felt when the arm is not used, and during use the pain is confined to the arm itself; but by and by, if efforts be persisted in, the sense of fatigue becomes more or less permanent, and extends upward from the arm, and may often be felt as a distinct pain between the shoulders. During all this time the power of the muscles for coarse work is in most cases not sensibly impaired, but the execution of any form of fine work is usually interfered with.

Even in the paralytic form of writer's cramp there is a certain amount of irregular spasmodic contraction of the muscles during the act of writing, as is especially shown by the stiffness and, occasionally, by the cramp of the fingers around the pen; but in the spasmodic form of the affection irregular muscular contractions are the dominant symptom. At first there are only simple, slight spasmodic movements of the thumb and first finger, so as to produce an irregular stroke in the writing, but after a time the spasms become stronger and more wide-spread. By a sudden extension of the finger the pen is dropped, or by a spasmodic action of the opponens pollicis with abduction and coincident flexion of the index finger the pen is rapidly moved from the paper, or occasionally a violent spasmodic flexion of all the concerned fingers holds the pen as in a vice. In extreme cases all the muscles of the forearm are involved; and it is asserted that the muscles of the arm and shoulder may be affected, although I have never seen an instance of this.

Much the rarest form of writer's cramp is that in which tremors are the most prominent manifestation. When any attempt to write is made, tremblings in the hand and forearm, and in extreme instances in the arm itself, come on. The pen, following the tremors rather than the effort of the will, soon makes nothing but

irregular undulating or angular strokes, in which not even the vestige of a letter can be made out. I have never seen a case in which tremors existed as the sole symptom, but I have seen them very marked in the spasmodic form of telegrapher's cramp, and have noted their persistence during almost all forms of voluntary movement, even after the occupation had been abandoned for months.

The symptoms of writer's cramp naturally lead to the supposition that it is a peripheral affection; but that it is intimately connected with a disordered condition of the nerve-centres is indicated by the fact that when the victim attempts to substitute the left hand for the right the cramp appears in that member also; and also by the circumstance, which I have repeatedly noticed, that it may be the first symptom of a general break-down.

Cortical Spasms.—Any of the muscles of the extremities or of the trunk may be affected with a local spasm. Such spasms may be due to disease of the cerebral cortex. The nature of such attacks is to be recognized by the occasional occurrence of Jacksonian epilepsy, or by the presence of other indications of disease of the cerebrum.

Hysterical Spasms.—Localized spasms are frequently hysterical. The nature of such a spasm is to be recognized by its apparent causelessness, by its sudden onset and departure, and by the presence of other hysterical manifestations, and the absence of evidence of organic disease of the brain, spinal cord, or nerves.

Inflammatory Spasms.—Localized spinal meningitis and spinal tumors, by irritating nerve-roots, may give rise to tonic and clonic spasm of the muscles tributary to such nerve-roots. The cause of such contractions is to be recognized by the presence of pain and vertebral soreness, either upon direct or indirect pressure, or of other symptoms of disease of the spinal membrane or of the vertebræ. Again, rheumatic contractions of the muscles may be mistaken for true local spasms: the diagnosis under these circumstances is to be made out by observing the presence of excessive pain upon passive or active motion, tenderness upon pressure, aching pains in the part when at rest, and other evidences of rheumatic disease, either in the present or the past history of the case.

Reflex Spasms.—Sometimes local spasm is of reflex origin. The recognition of these reflex spasms is often a very important

aid to the practitioner in diagnosing subacute disease of the joints or of the vertebral column. In any case of suspected joint or vertebral disease, a close examination should be made as to the power of motion in the part: thus, the patient in whom incipient caries of the spinal cord is suspected should be stripped, stood up with the feet close together, and then required to bend forward, backward, and laterally as far as possible. If it be found that the muscles of the back are thrown into spasm by any of these movements, the existence of local disease of the bone is very probable. The presence of the muscular spasm can sometimes be made out when otherwise it might be overlooked, by noticing that the movements in some one direction are very much more restricted than is normal.

Apparently Causeless Spasms.—A localized spasm in the trunk or extremities which is not hysterical, and for which no definite cause can be assigned, should, if persistent, be viewed with great suspicion, as it may be a manifestation of a hidden incipient centric disorder. There are, however, cases in which no cause for a local spasm can be made out, although the spasm may be absolutely intractable to treatment. Thus, I have seen a robust man, without discoverable disease of the genital or other organs, and without history of sexual or other excesses, in whom the testicles were frequently drawn up by spasm of the cremaster muscle with such force as to cause sickness of the stomach and syncopal sensations from the violence of the pain.

I shall not occupy space with details of the various distortions or irregular movements produced by spasms of the extremities. The reader who is desirous of tracing a spasm in any individual case to the affected nerve and muscle is referred to the pages upon local palsies.

A spasm, of course, causes phenomena which are the reverse of those produced by the corresponding palsy: thus, a spasm which produces abduction of the hand is due to an irritative lesion of the muscle and nerve, whose palsy causes loss of the power of abduction.

There are, however, certain muscular territories belonging to cerebral nerves in which spasm is so frequent, so severe, and so hidden in its causes as to require special notice.

Facial Nerve Spasm.—First among these cephalic spasmodic affections is that which affects the distribution of the facial nerve. When spasm of the muscles of the face is accompanied by pain, it is spoken of as *tic douloureux;* when there is no pain, the spasm is known simply as *tic.*

The contractions of a tic may affect all the muscles of expression, or may be limited to a very few of them. In the violent type of the disorder there is an incessantly repeated clonic spasm of the muscles of one side of the face, causing, in perpetual succession and alternation, winking, wrinkling of the forehead, movements of the nose, and even of the ears, drawing upward and downward of the angle of the mouth, etc. Usually the convulsive movements occur in paroxysms, lasting from a few seconds to as many minutes, then gradually subsiding into quiet, which persists for a greater or less length of time. Sometimes the periods of relaxation are very brief, or may seem altogether wanting. The paroxysms vary in number from two or three in the twenty-four hours up to thirty or even forty an hour. They are usually mild during the night, and sometimes disappear entirely when the patient sleeps.

Grasset asserts that, according to Jaccoud, such cessation is a proof of the reflex origin of the spasm; but I believe that this is not correct. Although almost any of the muscles supplied by the facial nerves may be affected, the spasms are especially prone to attack the orbicularis palpebrarum, the levator labii superioris alæque nasi, the zygomatici, and the corrugator supercilii: more rarely the frontalis or the platysma, and still less frequently the muscles of the ear, are attacked. The stylo-hyoid, the digastric, and the velum palati are very rarely, if ever, affected.

In some cases (in my experience especially when the attack is hysterical) the motor disturbance involves the various neighboring nerves. If the motor root of the trigeminus sympathize, the muscles of mastication are violently convulsed, so that the jaws are jammed together; and if at the same time there exists a unilateral spasm of the pterygoids, the teeth are violently ground on one another. If the hypoglossal nerve is affected, the tongue is thrust in and out of the mouth, and may be caught and severely bitten.

In a proportion of the cases of facial spasm certain points can

be found, pressure upon which will immediately cause cessation of the spasm. These points usually correspond to the situation of the Valleix points in trigeminal neuralgia. They may occur in every branch of the trigeminus, on the skin of the face, and in the cavity of the mouth. Remak has called attention to the fact that pressure upon, or galvanic irritation over, the transverse processes of the cervical spinal column will sometimes arrest the spasm. Occasionally these pressure-points, which should be diligently searched for, may be found in more remote parts of the body. The action of pressure upon the points of arrest is much more pronounced in blepharospasm than in the more diffused cases. Under these circumstances the eyes will frequently fly open as though a spring had been touched liberating a shutter.

The facial convulsions are in some cases limited to isolated muscles; the orbicularis palpebrarum is especially prone to suffer, giving rise to the affection known as *blepharospasm*, whose history was so elaborated by Von Graefe. The contraction is tonic, causing a complete closure of the eye, and consequent blindness. This is accompanied by innumerable bizarre grimaces, due to the efforts of the antagonistic muscles to overcome the force which is closing the lids. The tonic spasm may last for but a few moments, or it may continue almost without relaxation for weeks. It is prone to be excited by sudden exposure to light, by loud noises, or by any emotion. Blepharospasm is usually coincident with photophobia, and is generally reflex, due to some local inflammation of the eye, or more rarely to carious teeth, to ulcerations in the mouth or throat, or to some other local irritation at a point distant from the eye.

Another form of local spasm of the orbicularis oculi is the so-called *nictitating spasm*. It differs from blepharospasm chiefly in being clonic, so that the eye is rapidly opened and shut, instead of being held firmly closed.

Vaso-motor and trophic changes very rarely, if ever, accompany facial spasm.

Tonic spasm of all the facial muscles is spoken of by some writers, but in the majority of cases such alleged tonic spasm has, in all probability, been due to contractures following paralysis. (See Contractures.)

In any case of facial spasm it is the duty of the practitioner to

endeavor to discover the cause. It may be, first, reflex; secondly, rheumatic; thirdly, due to hysteria, or to a general neurotic condition of the system; fourthly, the expression of a centric disease. The reflex spasm has been noted as being produced by facial surgical traumatisms, by tumors and disease of the bone, by enlarged lymphatic glands, parotid abscesses, diseases of the teeth and jaw, and various irritations of more distant portions of the body, such as uterine disease, intestinal worms, etc. In many of these cases the spasm should perhaps be considered as due to a direct irritation of the facial nerve rather than as reflex. The nature of a reflex facial spasm is to be recognized by finding the point of irritation and noting the effect of its removal.

I have used the term rheumatic to cover the class of cases in which the spasm is precipitated by exposure to cold and wet. It is probable that under these circumstances there is a neuritis, and that the nerve-trunk would be found sensitive on pressure. I have, however, no personal evidence to offer on this point, and have not been able to find any in literature.

The hysterical form of the disorder is to be recognized by the presence of a neurotic or hysterical temperament and the absence of other cause. As already stated, this form of the spasm is particularly apt to involve contiguous muscles, and it is especially characteristic that at times spasms of these muscles should replace those of the facial territory.

Spasm of the facial nerve due to centric disease is the result of an irritating lesion existing either in the neighborhood of the facial nucleus or in that of the nerve-trunk. It is especially apt to occur in syphilitic disease. The serious nature of organic facial spasm is usually recognized without difficulty by its being associated with occasional epileptic attacks, or with other evidences of cerebral implication. Not rarely the centric facial spasm ought to be looked on as part of a Jacksonian epilepsy.

Finally, there is a residuum of cases of facial spasm in which no cause can be assigned for the spasm. Under these circumstances there is probably some degeneration of the facial nucleus.

Spinal Accessory Spasm.—Spasm of the muscles supplied by the spinal accessory nerve constitutes the *Tic rotatoire* of French and the *Tic-krampf* of German writers, and is not extremely rare. In the majority of cases it is unilateral, but not

infrequently it is bilateral, and implicates the muscles of each side of the neck. The sterno-cleido-mastoid muscle may be affected either alone or as a co-sufferer with the trapezius muscle. By the contraction of the sterno-cleido-mastoid of one side the occiput is drawn backward and towards the affected muscle, so that the chin is thrown upward and towards the normal side. At the same time the head is bent over so that the ear is brought nearer the clavicle. When the trapezius is alone affected, the head is drawn backward and towards the contracted muscle without rotation of the chin, whilst the shoulder is raised and the scapula brought nearer to the vertebral column.

Contraction of the trapezius without implication of the sterno-cleido-mastoid is unusual, but it is common for the trapezius to escape in part or entirely. When the muscles are simultaneously contracted, the movements produced by each of them are combined in various proportions, according as one muscle or the other is more violently affected.

The spasms come on in frequently-repeated paroxysms, which are often frightful in their violence. They usually cease during sleep, and are intensified by emotion or any kind of disturbance. In the severest cases the patient is disabled during the paroxysm from talking or performing any action. There is almost invariably more or less suffering during the paroxysm, and in some cases the pain is terrible. It seems to me that there are as clearly two forms of tic rotatoire, a painful and a non-painful spinal accessory spasm, as there are of tic. It is very rare to find points upon which pressure will arrest the paroxysm; on the other hand, I have noted painful points, pressure upon which induced paroxysms of horrible intensity. There is a very distinct tendency for the spinal accessory spasm to overflow into the spinal cervical region; and in a case in which I had both spinal accessory nerves cut the convulsions continued, although in a much modified form, evidently through the spinal nerves.

The causes of spinal accessory spasm are usually extremely recondite. It probably may be reflex, and it certainly may be hysterical, but in the great majority of cases no point of irritation can be found, and no evidences of centric disease can be discovered. The extreme obstinacy of the affection indicates, however, that it is due to some obscure degeneration of the nerve-centres.

Wry-Neck.—Tonic spasm of the sterno-cleido-mastoid muscle, and consequent fixed torticollis, or wry-neck, is usually rheumatic.

I have seen a severe torticollis produced in children by enlargement of the cervical glands, probably as the result of a direct irritation of the nerves. In some of these cases care is necessary to avoid being misled into believing that a centric disease exists, because the pupil upon the diseased side may be affected by pressure upon, or by irritation of, the sympathetic nerve fibres, which accompany the carotid artery and its branches through the skull and orbit into the eye.

TREMORS.

Tremors may be defined to be involuntary oscillatory movements which are produced by somewhat rhythmical alternate contractions of antagonistic muscles and do not prevent voluntary actions. They are normally present, to a certain degree, in many neurotic persons, in whom they are increased by excessive mental or physical work, by the free use of tobacco, coffee, or tea, and by any other action or agency which tends to increase "nervousness."

Pathological tremors may be due to certain poisons, to the alterations of old age, to the so-called Parkinson's disease or paralysis agitans, to multiple cerebro-spinal sclerosis, to general paralysis of the insane, and, in rare cases, to focal diseases of the brain.

Senile Tremors.—Senile tremors usually are developed at an advanced age, although in some cases they are manifested in early middle life, particularly under an hereditary influence. Their ordinary development is gradual. In most cases they are first seen in the muscles of the neck, or in the arms, from which they slowly spread to other portions of the body. During absolute repose they are naturally absent; but even the effort of extending or supporting a limb causes them to reappear. They are usually increased by excitement, mental or physical. The most characteristic symptom is the oscillation of the head, which is often accompanied by tremblings of the tongue and of the lower jaw. In advanced cases the muscles of respiration participate so that the speech is affected. Senile tremors are usually accompanied by a very gradual failure of muscular power, but there is no true palsy.

Toxic Tremors.—According to Lafont, *lead-poisoning* is sometimes accompanied by tremors, whose origin is indicated by their

being much worse at the end of the day. It is affirmed that in some cases these saturnine tremors are exceedingly violent and acute. Muscular contractility is said to remain intact.

Tremors are said also to be a marked phenomenon in chronic *mercurial poisoning*. Hallopeau states that in workers in mercury they are a very constant symptom, and are transmitted to the children. They usually begin as a very fine tremor of the lips, tongue, and extremities, gradually becoming more intense until they are excessive. In some cases they have come on suddenly. During repose the affected limbs are quiet, but the moment any attempt is made towards voluntary movement the tremors appear: so that there is a simulation of multiple cerebro-spinal sclerosis. The tremulousness of the tongue is said to be exceedingly constant, and to produce a peculiar staccato and hesitating speech. The head is quiet until very late in the disorder. There is usually insomnia; sometimes there are true clonic convulsions. The diagnosis must rest chiefly upon a knowledge of exposure to mercurial vapors.

Tremors are constant and characteristic in *chronic alcoholism*. They resemble somewhat those of old age, but their true nature is revealed by their being markedly worse in the early morning; by their being increased by abstinence from drink and quieted by a potation; and by their accompanying other symptoms of chronic alcoholism. (See page 27.) They are commonly worse in the hands than in any other portion of the body. In old drunkards the alcoholic tremor merges insensibly into the phenomena of senility.

Paralysis Agitans.—The characteristic phenomena of paralysis agitans, or Parkinson's disease, are tremors, progressive failure of power in the affected muscles, slowly-developed moderate rigidity, and, in the most advanced stages, peculiar alterations in the habitual positions of the body and in the gait.

Paralysis agitans usually comes on insidiously and gradually, although in some cases the symptoms have developed at once after a sudden fright or other emotional storm. The attention of the patient is first attracted by a tremor in the hand or foot, or even in one finger or toe. This tremor at first is transitory, can be controlled, at least temporarily, by an effort of the will, and is suspended by voluntary movement. Little by little, without any fixed method of progression, it involves more and more of the

body, becomes more and more settled, and at last continues throughout all the waking hours, during repose as well as during action, and cannot be controlled at all by the will. It often passes up the arm first invaded, and then descends to the lower limb of the same side, constituting the hemiplegic form; or it may commence in a leg and pass across the body to the opposite leg, and produce a paraplegic variety. Finally, all portions of the body are affected except the head. The face is very rarely attacked by the tremors, although in the later stages it puts on a peculiar fixed, immovable, usually melancholic expression. According to Charcot, the head is never affected,—any apparent trembling of it being due to the transmission of motion from the trunk. This absolute assertion is, however, not correct, as I have seen typical cases of paralysis agitans in which the muscles of the neck and the head were in constant tremor; and Westphal (*Charité Ann.*, 1877, p. 405) is said to have reported similar cases. Loss of power in the lips seems to be not infrequent in the advanced stages, so that there is a tendency to dribbling of the saliva, a tendency which is also in part due to the peculiar prone position of the head. The speech becomes a little slow and labored, but is not profoundly affected: neither eating nor swallowing is interfered with.

The tremors themselves are short, very rapid, and in some cases distinctly rhythmical, especially in the fingers, where they may assume somewhat the appearance of voluntary actions, as though the patient were rolling something between the digits. I have noticed in some cases a distinct tendency of the tremors to alter their rapidity in accordance with any rhythmical sound, so that their rapidity could be regulated, without the patient's being conscious of it, by altering the rate of vibration in the interrupter of a faradic battery. A peculiar rigidity of the muscles is characteristic of the advanced stages. There are no violent contractures, but a characteristic fixation of the part. To this statue-like rigidity is, at least in some measure, due the position of the patient. In standing the trunk is inclined forward, with the face looking obliquely downward; the forearms usually flexed somewhat upon the arms; the hands a little bent upon the forearms, and the fingers partially closed, so that the hands assume a position similar to that in which the pen is held: hence the term of "writing hand" as given

by Charcot. The same tendency to flexion of the legs exists, so that in standing the knees are bent. Occasionally, peculiar distortions of the hands or other portions of the body may be met with. On attempting to restore the normal position of the parts, the muscle usually offers but little resistance until the restoration is nearly perfected.

The power of making momentary muscular efforts diminishes very slowly in paralysis agitans, but even early in the disease fatigue follows moderate exertion, so that there is soon a great loss of endurance. In not rare cases there is a marked tendency to *festination* in the walk,—*i.e.*, to a progressive increase in the rapidity of the gait. The man seems to be in continual danger of falling forward when attempting to walk, so that the leg has to be thrust forward more and more quickly in order to prevent toppling over, and the walk becomes more and more rapid, and in a little while breaks into a run, which grows faster and faster, until the patient either falls or arrests his course by seizing hold of some stationary object. The peculiar position of the body would appear to be the cause of the accelerated gait, the head being thrown so far forward as to bring the centre of gravity beyond the line of the feet. That the festination depends upon something more than this is, however, shown by the fact that there are cases in which the tendency is to run backward instead of forward. Moreover, a very markedly bent position is not incompatible with a normal gait.

Sensation is not profoundly affected, and in some cases there is very little suffering. Usually, however, especially as the disease advances, there is a perpetual sense of fatigue in the affected muscles, which may amount to a severe aching. Very frequently the patient complains of an habitual feeling of excessive heat, which also may be manifested by a continual sweating. This sensation of heat does not depend upon any elevation of the central bodily temperature, which is of normal intensity. The studies of Grasset and Apollinario, however, indicate that there is an elevation of the temperature of the external surface of the body. These observers found that whilst the temperature of the surface of the forearm in the normal individual was 33.6° C., in a case of paralysis agitans placed under similar conditions of clothing and exposure the temperature was 36.8° C.

The urine has been chemically analyzed by Regnard (*Progrès Méd.*, 1877), who found the urea normal, the sulphates less than normal. According to Cheron (*Progrès Méd.*, 1877, No. 48), there is a constant increase in the quantity of the phosphates, which is characteristic, and may even precede the development of the tremors. This important observation needs confirmation.

The course of paralysis agitans requires many years for its full development, but if the patient does not die of an intercurrent disorder he passes into a condition of hypochondriasis, great depression of spirits, loss of intellectual power, general failure of nutrition, marked emaciation, loss of digestive power, and general marasmus, and at last dies of exhaustion, the end often being hastened by bed-sores or other local ailments.

Multiple Cerebro-spinal Sclerosis.—The tremors which are present in multiple cerebro-spinal sclerosis are characterized by their complete absence not only during sleep, but also during repose. In most cases the quiet sitting with the hands in the lap suffices to put aside all trembling, but in other instances it is necessary to put the patient to bed in order to get a muscular rest sufficiently absolute to allow complete cessation of the tremors. When any movement is made the tremors appear first in the part that is in action, but in most cases they in a little time spread throughout the body, so that the simple effort of writing may produce tremblings in every part of the organism. In contradistinction to ordinary cases of paralysis agitans, the tremors especially affect the head. They are always associated with a more or less pronounced palsy of the affected part.

In most cases there are some indications of disturbance of cerebration, such as loss of memory, or of the power of fixing the attention. As the symptoms depend upon the existence of isolated patches of chronic inflammation or sclerosis in the brain and spinal cord, it is apparent that the cerebral and spinal symptoms which accompany the tremors must vary almost indefinitely according as the exact seat of the sclerotic patches varies. The degree of mental impairment is in direct proportion to the amount of invasion of the upper brain-centres. Mental symptoms may be very slight, or even altogether absent, but hallucinations and other symptoms of insanity have been noticed. The usual tendency is, however, towards failure of the mental powers, or even com-

plete amentia, rather than towards active insanity. Charcot states that in about three-fourths of the cases of cerebro-spinal sclerosis vertigo is present. Usually objects seem to be whirling around with great rapidity, and the individual himself feels as though he were revolving. Not rarely the vertigo is so severe that the patient has to lay hold of something in order to maintain the standing position. Closely allied to the vertigo are the *apoplectic* attacks, which are in advanced cases quite frequent. These attacks usually come on suddenly without aura or other warning. Sometimes there is complete loss of consciousness, in other cases there are convulsive seizures, which may resemble those of major epilepsy. Commonly the patient recovers rapidly from such attacks, but occasionally a partial hemiplegia is left for a few hours, or even a few days. Death may take place during a paroxysm, when no lesion will be found in the brain to account for the acute symptoms. At the time of the attack the pulse is usually accelerated, and, according to the researches of Westphal, there is a rapid and characteristic rise of the central temperature. In the hours following the first apoplectic invasion a temperature of 102° F. has been noticed, and twenty-four hours later 104° F., the patient finally recovering. It is asserted that when the temperature passes above 105° F. death almost inevitably occurs. Charcot affirms that the congestive attack of disseminated sclerosis can be diagnosed from a true apoplexy occurring in this or any other affection by paying attention to these temperatures,—in cerebral hemorrhage any rise of the bodily temperature being always preceded by a fall, which is wanting in congestive apoplexy.

Ocular symptoms are very frequently present in multiple cerebral sclerosis. Nystagmus has been noted in a number of cases. Diplopia occasionally exists; but amblyopia is a much more frequent and persistent symptom.

In many cases symptoms due to invasion of the pons or medulla are present. A peculiar enunciation is almost characteristic of the disease, the patient hesitating in the articulation, although not distinctly stammering, and having special trouble with the consonants *l*, *p*, and *g*. The words are pronounced in a low, hesitating manner, with a certain regularity of accent and pause, somewhat after the method of school-boys in reading Latin poetry: hence this peculiar speech has been spoken of as "scanning."

Tremulousness of the tongue, with wasting, has been noted in some instances: in other cases all the paralytic and atrophic symptoms of the so-called progressive bulbar palsy are present.

The spinal symptoms may simulate those of any form of chronic sclerosis, or may consist of a mass of commingled types. Thus, if the sclerosed patches happen to be in the posterior root-zones, the fulgurant pains, disturbances of co-ordination, loss of the knee-jerk, and the other symptoms belonging to locomotor ataxia may be present. When the lateral columns of the cord are involved, contractures of the muscles with resultant deformities, exaggerated reflexes, and the other symptoms of lateral sclerosis may be well developed. If the patches have involved both the lateral columns and the contiguous gray matter, the symptoms resemble those of amyotrophic lateral sclerosis. Muscular contractures, heightened reflexes, and wasting of the affected muscles are present. The gait varies according to the spinal distribution of the sclerosed foci. It may be that of locomotor ataxia, it may be that of lateral sclerosis, or it may be a grotesque combination of the two.

The course of multiple cerebro-spinal sclerosis is usually slow. Five, six, or even eight years may be required for the wearing out of the prodromes. The intellectual disturbance finally deepens into dementia, the general loss of power into profound paralysis, the difficulty of speech into an unintelligible grunting, the muscular wasting into excessive trophic disturbances, with abscesses, ulcerative destruction of the internal mucous coats, and perhaps death from septicæmia. In the great majority of cases, however, the patient perishes of some intercurrent disease, especially of pneumonia, phthisis, or dysentery.

The diagnosis of multiple cerebro-spinal sclerosis is generally to be based upon the appearance of tremors during action and the slow failure of muscular power, since years may elapse before the occurrence of any other symptoms. When the patches of sclerosis are confined to the spinal cord there are no tremors: *i.e.*, in *multiple spinal sclerosis* tremors are not present.

CHOREA.

Choreic movements may be defined to be irregular movements produced by independent contractions of single or associated

groups of muscles not vibratory in character, and more or less simulating purposive movements, but never forming a complicated series of apparently purposive actions. They may vary in intensity from the slightest, irregular movements of the fingers or toes, or even a mere condition of excessive muscular activity resembling restlessness, up to the most severe and violent motions. They may be confined to a single group of muscles, under which circumstances they may be considered as forms of local spasm, or they may involve associated groups of muscles, or the entire muscular system of the organism. When, however, the whole body is affected, the muscular contractions do not take place regularly or consentaneously, but momentarily here and there. They are often under the control of the will for a short period of time, but always assert themselves in a few minutes, and in many cases cannot be controlled at all.

The choreic movement is usually irregular and not at all rhythmical, but in some cases is more or less regular, and it may be entirely rhythmical. Rhythmical choreas more or less closely resemble tremors, differing chiefly in that the movements are much slower and more extensive.

When the choreic movements involve all parts of the body the patient may be said to be suffering from general chorea.* When

* The names of St. Vitus's Dance, the Dance of St. John, Chorea Minor and Chorea Major, and Chorea Germanorum, have been so much used and with such different meanings that it seems necessary to say a few words here in regard to the signification with which they will be employed in this book. It appears that the Phrygian Bacchantes, in their wild worship, were affected with furious uncontrollable automatic movements, accompanied by more or less disturbance of consciousness, and it is certain that the sect of the Suffi, in Persia, shortly after the origin of Mohammedanism, were accustomed in their sacred ceremonies to pass into a condition of raging excitement, with furious dancing, convulsive tremblings, and even general convulsions. About the year 1000 a sect of the Suffi found numerous followers and imitators throughout Asia Minor, Persia, and Egypt, and even in Greece. In Christian lands the so-called Dance of St. John was already at the time of the Crusades an observed custom; and when the influence of the Suffi spread itself by the returning waves of the Crusades, the epidemics of religious excitement and automatic dancing became more and more violent. It was not until the outbreak, in 1418, of a fresh epidemic in Strassburg, that the term Dance of St. Veit began to be freely applied to these religious disorders, a name which appears to have had its origin largely in the fact that in these later epidemics children were especially affected. St. Veit was a boy who, born in

the choreic movements are fixed in one part, the term local chorea may be used.

GENERAL CHOREA.

Acute general chorea is usually due to St. Vitus's dance.

St. Vitus's Dance.—St. Vitus's dance is a non-febrile disease, generally occurring in children, which is characterized by the presence of choreic movements usually involving all portions of the body, although liable to affect especially one extremity or one half of the body, associated with a condition of general lack of tone, and often with a distinct loss of muscular power. The invasion of this disease may be sudden or gradual. The attack may come on in the midst of apparent health, but ordinarily it is preceded by languor, irregular action of the gastro-intestinal tract, and a pronounced nervous irritability. The motor disturbance may be first indicated by a peculiar restlessness of the child, who is not rarely punished for fidgeting. The true choreic movements usually appear first in the fingers, and shortly afterwards in the face, and spread until they involve the whole body. In severe attacks the arms are in almost constant movement, the fingers opening and closing, the wrists flexing and extending, and the elbow-joints in almost incessant activity, so that every imaginable position of the hand and arm is rapidly taken and lost. During the violence of the disease it is impossible for the child to control the movements of the arm sufficiently to dress or feed himself, or to perform

the island of Sicily, suffered martyrdom in the year 303 during the persecution of Diocletian, and whose body, carried hither and thither for a considerable length of time, found its final resting-place in the cloister of Korvey.

By Paracelsus these epidemics were called Chorea Sancti Viti, and Chorea Lasciva. The disease of childhood now known as St. Vitus's dance has no connection either etiologically or in its nature with these epidemics, but modern custom enforces the application of the name to it as used in this book. By many German writers the affection of childhood is known as Chorea minor, whilst the term Chorea major, or Chorea Germanorum, is used to express affections more or less closely resembling in their phenomena those of the epidemic furies of the Middle Ages. By some German writers any very bad case of ordinary chorea is spoken of as Chorea magna. In the present work the term Chorea major is used with its more limited signification, as expressing a disease in which occur paroxysms of movements that are automatic and beyond the immediate control of the will of the patient, although closely resembling voluntary acts in their apparent purposiveness and in their sequence.

any act requiring precision of motion. At this time the legs are similarly affected, so that walking is gradually interfered with, or may be rendered impossible. The steps are irregular, jerking, often with lateral movements, now rapid, now slow, and if progression occur at all it is zigzag and uncertain. The face and head are no less affected: there is a constant, ever-changing distortion of the countenance, giving rise to fleeting expressions of sadness, terror, grief, rage, etc., and to grimaces innumerable. The mouth is opened and shut, the corners jerking up and down: the tongue is protruded, or sometimes moved rapidly in the mouth so as to produce a peculiar clacking sound. Articulation grows indistinct, the child speaks irregularly and badly, perhaps only in monosyllables, and finally the voice may be converted into a succession of irregular unintelligible sounds. In very bad cases mastication becomes almost impossible, and even the muscles of deglutition are involved, so that the child is unable to swallow at the proper moment, and the food is spluttered and spilled about. The head itself is moved rapidly to and fro, backward and forward, sometimes laterally, sometimes in perpetual rotation. In the most violent cases all the muscles of the body are in a condition of furious action. The rolling, twisting movement of the trunk, and the perpetual beatings and thrashings of the extremities, render it almost impossible for the patient to lie in bed unless fastened down, and the utmost care is necessary to prevent severe bruises and excoriations of the skin.

The respiratory muscles are the last to be affected, but cases have been reported in which hiccough, crowing inspiration, irregular respiratory rhythm, and other evidences of choreic action of the respiratory muscles were abundantly present. The choreic movements cease at night, or at least during sleep, but in the most severe cases by keeping the patient awake they produce an insomnia which constitutes an additional factor in the rapid wearing out of the strength and the bringing about of a fatal result. That the brain-cortex does not entirely escape is shown by the peculiar nervous irritability which forms an almost essential symptom of the disease. The general intelligence is ordinarily well preserved, but there can often be noted a temporary weakness of memory, and the loss of the power of fixing the attention upon any one subject for a length of time is usually very decided.

Hallucinations are very rare, and usually indicate that a chorea is hysterical. They may, however, occur in typical St. Vitus's dance. In fatal cases the mental disturbances are very pronounced; there may be even an acute dementia: sometimes the patient is seized with maniacal delirium, which is always of exceedingly serious import.

The muscles of organic life may participate in the choreic disturbance. This is especially true of the heart. Chronic valvular lesions are frequent among choreic patients, and an acute endocarditis occasionally occurs during an attack of St. Vitus's dance; but cases are not rare in which mitral or even aortic murmurs are heard during an attack which are not due to any organic lesion of the heart and are not hæmic in their origin. This is shown by the fact that these murmurs occur when there is no anæmia, that they vary from day to day and from hour to hour and at times may be absent, and that when the child recovers from the chorea the murmur disappears entirely.

Further, fatal cases have been reported in which no valvular lesion was found at the autopsy, although marked cardiac murmurs had existed during life. (See *Revue mens. des Maladies de l'Enfance*, 1884, ii. 421.) The most rational explanation of these murmurs is that they are due to the irregular contractions of the chordæ tendineæ preventing the proper closure of the valves. It is the duty of the practitioner always to auscult the heart of the choreic child, and if murmurs be present to decide, if possible, their significance. If the history of a previous endocarditis or of previous chronic valvular lesions can be obtained, the probabilities are always that the murmur is due to an old lesion. The absence of such history is, unfortunately, no proof of the previous non-existence of cardiac disease. Supposing that the murmur is recent, it is often a very difficult matter to decide whether it is neurotic or inflammatory. The neurotic murmur rarely, if ever, manifests itself in irregularity of the pulse; it is not associated with cardiac pain, nor with elevation of the general temperature. If these exist, the diagnosis of acute endocarditis may be considered made out. The presence of even one of these symptoms should lead the practitioner to treat the case as one of endocarditis.

Whenever a cardiac murmur is heard in a choreic patient, unless its nature be very apparent great care should be exercised in the treatment of the case, and a guarded prognosis should be given, because a murmur which is apparently neurotic may fail to disappear after the child's recovery, and because the rapid and complete disappearance of a murmur which was apparently organic may prove it to have been neurotic.

Nature and Limitations of Chorea.—The St. Vitus's dance or chorea of childhood is a very frequent disorder, having, as already stated, clearly-defined clinical characteristics, and would seem, therefore, to deserve a distinct place in nosology.* Choreic movements may, however, be produced by peripheral irritations, and in some cases these movements have been universal, and so severe as even to threaten life. Dr. C. Fischer reports (*Zeitschrift für Wundärzte*, 1853, vol. vi. p. 89) the case of a young peasant girl, in whom a futile attempt to remove a tooth was followed by the formation of an abscess, and by marked unilateral chorea, which lasted two years, until Dr. Fischer removed the roots of the tooth, when the movements ceased at once.

Dr. R. Fischer (*Oester. Med. Wochenschrift*, 1841, p. 46) reports a case in which a general chorea ceased at once upon the expulsion of a tapeworm. Dr. Edmond Cenier records a case similar to this in the *Gaz. Méd.-Chir. de Toulouse*, 1877, p. 43. The chorea was so violent as to threaten life, and had persisted several months, notwithstanding treatment. Amelioration began five days after the expulsion of the parasite, was very rapid, and resulted in complete cure. In the *Journ. de Méd. et de Chirurg.*,

* Dr. L. Rousse, in his *Thesis* (Paris, 1865, No. 252), affirms that in ordinary chorea of childhood points of pain, upon pressure, can be found in nerve-trunks where they become very superficial, or where the nerve-trunks emerge from bony places, or where the nerve-filaments traverse a muscle to reach the skin, or where the finer filaments come near the surface. The pain is stated to be quite severe. He asserts that the neuralgic points of Valleix are well marked, and he finds in the head four especial choreic pain points,—the occipital point just below the occiput, and cervical superficial points in the middle of the neck, a little below the middle of the neck, and upon the posterior edge of the sterno-cleido-mastoid, corresponding to the mastoid point upon the mastoid process. He further gives a large number of points found in connection with other portions of the body, and corresponding to the generalizations which he makes as detailed above.

Paris, 1841, is recorded the immediate arrest of a chorea of one month's duration by the expulsion of lumbricoid worms.

M. Borelli reports (*Bulletin de la Soc. de Chirurgie*, 1852, p. 292) the case of a boy thirteen years old, in whom a violent chorea which had resisted all treatment for six months was cured by the removal of a neuromatous tumor from beneath the foot. The movements became less the day after the operation, and by the fourth day had ceased entirely. In the *Revue de Méd.*, 1884, p. 568, Dr. E. Weill reports a very interesting case in which a decided hemichorea was produced by intrapleural injections, the movements being upon the same side of the body as the pleurisy.

Further citations of cases might readily be made, and especially an abundance of opinion might be obtained from recognized authorities, showing the occasional dependence of chorea upon the presence of intestinal worms; but I think enough has been here said to prove the existence of a *reflex chorea*.

A large number of autopsies have been made upon children and adults dead of St. Vitus's dance, and the results have given rise to much discussion. In my opinion the older autopsies ought to be disregarded. The means of investigation were so imperfect, and were so imperfectly used, that the danger of being misled by these observations is greater than the chance of receiving enlightenment. Nevertheless, it seems to me that, after throwing overboard much rubbish, there remain certain cases in which, after properly-conducted autopsies, no appreciable lesion could be found. The positive results which have been reached in other cases comprise—first, brain-alteration; secondly, alteration of the spinal cord.

Among the most remarkable papers upon the brain-lesions of chorea is that of Dr. Broadbent, who has demonstrated that the corpus striatum and thalamus opticus are in some cases the location of the lesion. Dr. Broadbent states that a variety of morbid conditions of these ganglia may produce chorea, but the most frequent alteration in his cases was a capillary embolism of the corpus striatum, thalamus opticus, and their vicinage. A number of autopsies have been made confirming the existence of capillary embolism in the brain in fatal chorea, and it would seem as though there were an intimate relation between chorea and this condition of the brain. The association is too frequent and too peculiar to be

merely the result of chance. On the other hand, it is evident that in many cases of chorea no such lesion exists. It is absurd to suppose that the chorea which is produced in a few hours by a fright and is cured in a few days by arsenic is the result of so serious an organic lesion as that indicated. Moreover, there has been an abundance of autopsies in which capillary embolism did not exist. Again, as in the case reported by Tuckwell, other changes in the brain have been noted besides those of embolism.* It must, therefore, be concluded that an acute chorea may be intimately associated with minute cerebral embolism, and also with other lesions of the brain; among which lesions may be especially mentioned the peculiar alteration of the ganglionic cells of the brain noted by Meynert as pervading the whole organ in a case of chorea.

In regard to the spinal cord, the following paragraph from the article of Von Ziemssen in his Cyclopædia sums up the evidence to the date of its writing, 1877: "In the spinal cord alterations have been repeatedly found,—namely, hyperæmia of the medulla and the membranes, softening of the cervical and also of the dorsal medulla (Romberg, Ogle, Gray, Golgi, De Beauvais, Hine, Brown-Séquard, Lockhart-Clarke); interstitial proliferation of nuclei and hyperplasia (Rokitansky, Steiner, Meynert, Elis-

* In the cases collected by A. Foucherand (*La Chorée*, Lyons, 1883), inflammatory lesion of the central ganglia (opto-striate bodies), and periencephalitis with and without disease of the central ganglia, are represented. Dr. J. Muellendorff reports (*Deutsch. Archiv für Med.*, 1880, vol. xi. p. 609) a fatal case due to disease of the sphenoid bone, with compression and slight softening of the pons. Dr. Hughlings-Jackson details (*Brit. Med. Journ.*, 1875, vol. i. p. 636) a case in which the symptoms for a considerable time exactly resembled those of the St. Vitus's dance of childhood, and in which, after death, there was found a tuberculosis affecting the surface of the pons, medulla, cerebellum, and the whole base of the brain, also the island of Reil, and neighboring convolutions, which were softened, as well as the gyrus fornicatus. The posterior cerebral arteries were involved, but not the vessels of the corpora striata, and no emboli could be detected. Dr. H. M. Tuckwell reports (*St. Barth. Hosp. Report*, 1869, p. 87) a case of chorea in which, after death, there was found softening of the right cerebral middle lobe, involving the deeper layer of the cortex and the subjacent white substance, and extending into the neighborhood of the corpus striatum and optic thalamus. The corpus striatum was seemingly not affected; the optic thalamus was slightly affected in its upper and outer aspect. The corresponding region in the left hemisphere was likewise soft.

cher), and sometimes serous exudation in the central canal, proliferation of nuclei in the adventitia of the vessels, and regressive metamorphosis in the ganglion-cells (Elischer)." Since this paragraph was penned, spinal lesions have been found in chorea by Dr. Dickinson, by Dr. Bury, and by Dr. James Ross. According to Dr. Dickinson, the part of the cord especially affected in the disease is "the central portion of each lateral mass of gray matter comprising the root of each posterior horn."

Dr. Ross says of a case, "I was struck with the alteration presented by the accessory cells of the anterior gray horns: they appeared shrivelled, their protoplasm was granular, their nuclei were obscured, and many of their processes were indistinct or absent."

Without indulging in further quotations, it may be stated that we have the evidence of at least five or six different observers as to the alterations of the spinal ganglionic cells in acute human chorea, and that in numerous other cases these alterations in all probability existed, but were overlooked. I do not mean to assert that an appreciable lesion of the spinal cells is always to be found in St. Vitus's dance; indeed, I am confident that the disease may prove fatal without such lesion. Nevertheless, I am well assured that such alterations are very frequent in fatal cases, and that in not a few instances they have been overlooked because not appreciated and sought for.

There is a form of chorea which is not uncommon in the young of certain of our carnivorous domestic animals. I have never seen a case in herbivora; but Professor Huidekoper, of the Veterinary Department of the University of Pennsylvania, informs me that he has treated the disease in calves. The relations of *animal chorea* with the chorea of childhood have been considerably discussed, with difference of conclusions. Except in the case of contagious diseases, it is impossible to determine with absolute positiveness that a certain disease in the animal represents a certain disease in man; but to my mind it is plain that if canine chorea be not the same disorder as the St. Vitus's dance of childhood it is very closely allied to it.

It is true that the movements in canine chorea are usually rhythmical, whilst the movements in the child are ordinarily not so, and much has been made out of this difference. I have, however, seen dogs in which the movements were not rhythmical, but

had all the *gaucherie* of the chorea of childhood, and in some cases of children the choreic movements approximate the rhythmical type. The points of resemblance in the two affections are close and striking. In each disease it is the young that are especially attacked; in each the chief symptoms are those of disordered motion; each affection is connected with a constitutional disorder (rheumatism in the child, and distemper in the dog); in each the movements can be temporarily inhibited by the will, are not accompanied by disorder of sensation, and are associated with loss of power and lowered general nerve-tone; finally, in each disease arsenic is recognized as the standard remedy.

The important point in this matter is that it is proved (see paper by author, *Therapeutic Gazette*, May, 1885) that the movements in animal chorea originate in the spinal cord. My own studies of the spinal cords of choreic dogs have shown that the basal lesion is a peculiar condition of the ganglionic, or multipolar, cells. Gowers and Sankey have noted an infiltration of the gray matter with leucocytes, especially in the neighborhood of the vessels, and a similar condition has been seen by M. Pierret (Foucherand, *La Chorée*, Lyons, 1883), but, for reasons given in detail in my paper, it seems to me that the change in the cells is the main lesion, and in this I have been recently confirmed by Professor Horsley, of London. Further, this is in accord with the previous observation of Professor Putnam, of Boston.

When the animal was killed in the very beginning of the attack, the cells showed no change; a little later the only alterations in the cells were the very frequent absence of the nuclei, the failure of granulations in the protoplasm, the loss of power to take staining fluids, and rarely the occurrence of sharply-defined vacuoles. Then the processes began to drop off; and finally it was found that the places of the cells were occupied by irregular, globose, crumpled-looking masses, without sharp outline, and taking carmine staining very faintly. No granulations, no nuclei, no processes, were apparent. These masses represent the cells in the last stages of degeneration.

There is probably at first only a functional disturbance of the spinal cell. The distinction between functional and organic disease is a purely arbitrary one. Functional movements are the results of nutritive changes, and a functional disorder is one in

which the nutritive changes have occurred, but have not sufficiently advanced to be recognized by our comparatively gross methods of study. The structure of the ganglionic nerve-cells is so complex in its ultimate nature, and yet so simple in its microscopic appearances, that it may be permanently and very seriously altered without leaving a physical trace which we can recognize. The first change in canine chorea is an altered nutrition—*i.e.*, functional excitement (or depression)—of the multipolar cells of the spinal cord. This altered nutrition may continue until the structure of the cells is entirely destroyed, but it may never go beyond a condition of change so non-apparent as to be unappreciable to us. The studies of Dr. Putnam show that in the kitten the brain-cells suffer like those of the spinal cord, and it is most probable that in animal chorea the movements originate in the altered nutrition of the spinal cells, but that throughout the nervous system the ganglionic nerve-masses suffer, so that the disorder is really a condition affecting not merely the spinal but rather the whole nervous system.

The bearing of what has just been said upon the question as to the nature of St. Vitus's dance is very obvious.

The marked tendency of the choreic movements to affect one side of the body or one limb has led many observers to confine their careful examinations to the brain, this hemiplegic or monoplegic tendency being believed to show that the movements originate in the brain and not in the spinal cord. A clinical study of the various human affections of the motor ganglia of the cord shows, however, that in such disorders spinal monoplegias are not at all uncommon, and that in some cases hemiplegias may be seen. In many cases of chorea of the dog this localization of the movements in one limb or in one side of the body is quite pronounced; indeed, this was the case in dogs in which I positively determined the spinal cord to be the source of the movements: it cannot therefore be considered that a hemiplegic chorea, much less a monoplegic one, is necessarily of brain-origin.

The similarity of the lesions which have been recently noted in the ganglionic cells of the spinal cord in fatal cases of human chorea to those which I have found in the dog is very apparent, and increases the probability that the two affections are essentially the same disorder. Whether this be so or not,

it is proved that choreic movements in the dog may originate from a diseased condition of the ganglionic spinal cells, and it is contrary to all known physiological laws that what in this respect is true of the dog should not be true also of the human being. That a purely spinal chorea may exist in man is further shown not only by what has already been said, but also by the case reported by Foucherand, in which in a chronically choreic child the brain was found to be healthy but the cord full of minute inflammatory foci. Further, since a diseased condition of the spinal ganglionic cells has been found in several cases of St. Vitus's dance, it must be allowed that such diseased state is at least one of the fundamental pathological alterations of human chorea. It is perfectly clear, on the other hand, that the disorder is not confined to these multipolar cells. The researches of Dr. Putnam, already quoted, show that in the cat alterations may be found, at least in some cases, in the brain ganglionic cells, and every clinician knows that the cerebral functions are often profoundly affected in the choreic child. The will, the intellect, and the emotional faculties are all prone to show the presence of an abnormal influence in chorea; and it seems, therefore, that we must consider that in the *choreic child the ganglionic cells in the whole cerebro-spinal system suffer, and that this alteration is the base of the disease;* in other words, the *pathology of the St. Vitus's dance of childhood may be said to be a diseased condition of the ganglionic structures of the cerebro-spinal axis, which abnormal state may exist without alterations of structure sufficient to be determined by the microscope, or may go on until it is accompanied by marked structural lesions.* Further, this condition must be looked upon as one of lowered tone, and it must be allowed that it may be produced by various causes, but is not likely to occur in persons of robust nervous system. The vital choreic depression of the nerve-cells may be the result of emotional disturbance, as in the chorea produced by fright. It may be the result of the influence of the rheumatic diathesis or poison upon the affected tissues. To the greatly depressed condition of the spinal ganglionic cells is due the fact that in almost all severe cases of St. Vitus's dance in children some degree of general or local muscular weakness exists. This in many cases amounts to a partial palsy, which may take the hemiplegic, paraplegic, or monoplegic form. The paralysis is never complete

unless, indeed, the chorea be associated with, or dependent upon, some organic lesion of the nervous system, and it almost invariably is recovered from without difficulty as the choreic symptoms subside.

On the other hand, it is very certain that minute brain-embolisms and other lesions exclusively of the brain will produce a chorea. Not only does this rest upon an abundance of clinical evidence, but M. Raymond has produced chorea in dogs by injuring the posterior part of the foot of the corona radiata in the brain. It would appear, therefore, that *lesions of any of the ganglionic cells connected with the direct cerebral or pyramidal tract may cause chorea;* and that there are various forms of acute chorea, or, to state it more correctly, that there are various acute diseases in which chorea is a prominent symptom.

We must conclude that chorea is no more uniform in its significance than is paralysis, and that it may be due—

First, to the chorea of childhood, or St. Vitus's dance.

Secondly, to reflex irritation.

Thirdly, to organic disease of the nerve-centres.

Fourthly, to pregnancy.

Fifthly, to hysteria.

Sixthly, probably to conditions of the nerve-centres not as yet made out.

Sufficient has been said in regard to choreas of the first three classes.

Chorea of Pregnancy.—A form of chorea the immediate cause of which is uncertain is that which occasionally occurs during pregnancy. It is frequently a very severe affection, in which the movements are so excessively violent and incessant that they deprive the sufferer of sleep and rapidly cause a fatal exhaustion. There seems to be on the part of obstetric authorities a tendency to believe that this chorea is a reflex neurosis; but the clinical histories of the cases show that a remarkable proportion of the patients had suffered from chorea during childhood, that sometimes the symptoms are mild, closely resembling those of the St. Vitus's dance of childhood, and that they are often accompanied by the peculiar muscular weaknesses seen in that disorder. (Cases, Dr. Fehling, *Archiv für Gynaecol.*, 1874, vol. vi. p. 137.) Moreover, in a number of instances distinct organic lesions of the brain

or of the spinal cord have been found after death. These lesions have consisted of slight hyperæmia of the brain and very great effusion in the right ventricle (*Obstet. Journ. of Great Britain*, vol. iv. p. 80); of softening of the corpus callosum, medulla oblongata, and cerebellum (*Obstet. Trans.*, Lond., vol. x. p. 159); and of softening of the cord (*ibid.*, pp. 163, 164, etc.). Further, if the symptoms of chorea of pregnancy were purely reflex, removal of the fœtus should bring quiet. Yet in six of the seventeen fatal cases collected by Dr. Barnes (*Obstet. Trans.*, 1869) no effect was produced by emptying the uterus; also in the case reported by Dr. Goodell (*Amer. Journ. of Obstet.*, 1869) removal of the child was without influence. The facts that are at present known concerning the causation of chorea of pregnancy may be summed up in a single sentence. There are usually a predisposition to chorea, inherited or acquired, inanition of the nervous system incident to the hydræmic state of the blood during pregnancy, and various potential peripheral irritations, especially in connection with the sexual organs. The most rational explanation of the chorea of pregnancy is that it varies in its immediate pathology, the pregnancy simply producing a condition of the nervous system which predisposes it to be thrown into an active chorea by various exciting causes.

Hysterical Chorea.—A general or local chorea may be produced by hysteria. The movements may be limited to a single limb, or they may be hemiplegic or paraplegic, and not rarely they involve the whole body. They are often disorderly and irregular and closely simulate those of ordinary St. Vitus's dance. Under such circumstances their true nature is to be recognized by the existence of marked concomitant symptoms of hysteria, and especially by the occurrence of occasional or persistent rigidity of the affected muscles. The true choreic neurosis may, however, coexist with, or perhaps depend upon, the hysterical neurosis, so that it would often be equally correct to speak of a patient as suffering from hysterical chorea or from choreic hysteria.

It is especially in hysteria that the peculiar brusque, rapid muscular contractions occur to which the name *electric chorea* has been given by the French writers. (See *Thesis*, F. Colaneri, Paris, 1884; also A. Guertin, Paris, 1881.) In this disorder the whole body, or any portion of it, is the seat of more or less rapidly

repeated, violent muscular spasms, resembling those produced by a sudden severe electric shock. It is asserted that electric chorea may be a symptom of chronic alcoholism: thus, in the case reported by M. Landouzy (*Soc. de Biol.*, 1873, May 31), an habitual drunkard, aged thirty-seven, suffered from manifestations of this form. When the man was lying on his back the legs would be flexed upon the thigh, and the thigh upon the pelvis, with slight abduction, then suddenly would be violently extended with a rhythmical movement at the rate of sixty or sixty-five times a minute. Similar cases are on record; but I am inclined to believe that they are simply instances of hysteria occurring in persons who have abused alcohol.

Rhythmical Spasms.—Choreic movements of hysteria are very prone to take on the form of vibratory spasms and to become more or less rhythmical. The vibrations may be very rapid. They frequently attack extremities distorted by hysterical contractures. Thus, in a leg violently flexed by contractures I have seen the knees vibrate laterally over a considerable arc at the rate of one hundred and twenty times a minute. By tracing a series of cases it will be seen that disorderly choreic movements insensibly pass into vibrations, and these into true rhythmic spasms. Rhythmic spasms may affect any portion of the body. The limbs, normal or distorted by contractures, may be agitated with regular movements. The face may be attacked rhythmically, and facial grimaces, with or without the consentaneous thrusting forward of the tongue, occur. Occasionally the muscles of the larynx and of respiration are also affected, so that each spasm is accompanied by a quick, strange utterance. This rhythmic chorea again passes by insensible degrees into the purposive movements of hysteria: thus, the rhythmic movements of the legs may give rise, when the patient is standing, to a series of rapid changes of posture resembling the mazourka or other dance.

LOCAL CHOREAS.

Paralytic Chorea.—Of the various local choreas, I shall first speak of those which are connected with hemiplegia or monoplegia of cerebral origin. In some cases the movements precede the cerebral hemorrhage, constituting the so-called *pre-hemiplegic*

chorea. In other cases they come on after hemorrhage, and are spoken of as *post-hemiplegic chorea*. Not rarely they fail to develop until the paralysis is distinctly growing less, and, it may be, has almost entirely passed off. They may come on gradually or suddenly, and are usually most marked in the hand and arm, next in the face, and only in rare cases in the leg. The muscles which are employed in delicate and complicated movements are especially prone to be attacked. The interossei muscles of the hand are very frequently affected simultaneously with their associated muscles of the forearm. Almost every variety of motion may occur. Sometimes the fingers are rapidly opened and shut. Again they are in perpetual flexion or extension. The hand itself is often folded and opened out. The wrist may be alternately flexed and extended, the forearm pronated or supinated, and bent or straightened at the elbow; not rarely the whole arm swings with an incessant pendulum movement from the shoulder-joint.

In many cases the movements of paralytic chorea are incessant during the waking period, and cease only when the subject goes to sleep. Yet in not a few instances they can be partially controlled by placing the hand in some peculiar position: thus, in a case under my own care, when the arm was put behind the body partial quiet was obtained; and by fixing the hand against the front of the body immediately under the breast, the woman was able to control the movements sufficiently to do crocheting. In a case reported by Ross, putting the hand in the pocket was sufficient to obtain rest.

In some instances the contractions are much more marked during voluntary movement than during rest. Indeed, sometimes post-hemiplegic chorea is represented simply by a lack of power of co-ordination and control, so that during quiet there is no movement of the part, but whenever a voluntary act is attempted the muscles are thrown into irregular spasmodic action. As pointed out by Dr. S. Weir Mitchell, who appears to have been the first clinician to study post-paralytic chorea, there are some cases in which the movements simulate purposive acts. Thus, in one of Dr. Mitchell's cases the patient, after an attack of right hemiplegia, so incessantly rubbed at the right leg with the right hand as to wear out the pantaloons. In another case the arm was alternately pronated and supinated, and in a third the arm

was swung across the body only during walking, at each step the fingers being firmly flexed.

The movements of the face may affect the whole distribution of the facial nerve of the affected side, or may be located simply in certain parts of its territory. They give rise to all sorts of grimaces and disturbances of expression.

A very curious association of movements is sometimes seen in post-hemiplegic chorea: thus, in a case under my own care, whenever the woman winked a very peculiar spasm occurred, involving only a few fibres of the facial nerve, and causing a peculiar dimple in the chin. In this case the oculo-motor ganglion had become linked to a few cells of one facial nucleus, so that a simultaneous nervous discharge from two centres was provoked by one peripheral irritation, or by one effort of the will.

One form of local chorea, which is usually, if not always, connected with chronic brain-lesions (especially sclerosis after infantile cerebral hemorrhage), is that to which the name *athetosis* has been given. In this the fingers or toes continuously and slowly assume various distorted positions, and are only partially under the control of the will. The spasms of the muscles of the forearm change so slowly that they might well be described as slowly-shifting tonic spasms. Athetosis is simply a symptom, not a disease, and indeed as a symptom scarcely deserves a name, since it is only one of an innumerable variety of post-hemiplegic spasms, and is never exactly the same in two cases.

A condition which occasionally follows a cerebral paralysis, and to which the name of *hemiataxia* has been applied, may be considered simply as a very mild form of post-paralytic chorea. In this affection disorderly, irregular, spasmodic movements occur when voluntary actions are attempted, although there are no muscular contractions at other times. Speaking of such a case, Dr. Mitchell says, "This patient had no involuntary or spontaneous movements, no motor disturbance until voluntary acts were attempted, when they at once became irregular. Those of the hand were, as I recall them, so striking that they possessed every clinical peculiarity of the chorea of childhood." Other observers attribute the irregularity of movement to the loss of the power of co-ordination; but that this is probably not correct is shown by the fact noted by Dr. Mitchell, that the movements were as well performed

in the dark as in the light. (See also Gowers, *Medico-Chirurg. Trans.*, 1876, vol. lxx. p. 321; Grasset, *Progrès Méd.*, viii., 1880.) In Dr. Mitchell's case all the extremities seem to have been somewhat affected, but the right hand was the most so. At the autopsy there was found very pronounced general atheroma of the cranial blood-vessels, and a spot of softening in the right crus cerebri, also one in the left corpus striatum.

Very generally post-paralytic chorea is associated with a more or less marked disturbance of sensibility. This hemianæsthesia may, however, almost completely disappear, although the choreic movements continue as violent as ever. The clinical reports seem to show that hemianæsthesia is not so absolutely essential, even to the post-hemiplegic chorea of adults, as is stated by some writers.

The accumulating clinical records confirm the original supposition of Dr. Mitchell, that post-paralytic chorea is most frequent when the attack of hemiplegia comes on in early life: hence the disease is especially marked in children suffering from paralysis the result of sclerotic patches in the brain, such as has been fully discussed in the chapter on Palsy. (See p. 75.) It should, however, be distinctly understood that this form of local chorea may develop at any age.

The particular seat of the lesion in post-hemiplegic chorea, as first stated by Charcot, and especially developed in the thesis of his pupil, Raymond, is in the posterior part of the internal capsule, in the immediate neighborhood of the lenticular nucleus and optic thalamus. The immediate band of fibres of the corona radiata especially involved is in front of that connected with and covering the posterior end of the optic thalamus. This region, it will be remembered, is a very distinct one, having its own artery, the posterior optic. Although in many cases of post-hemiplegic chorea the lesion is located in the spot designated by the great French neurologist, such location is not invariable. M. Demange (*Rev. de Méd.*, March, 1883, p. 377), after reporting a case with Charcot's lesion, records one in which there was violent post-hemiplegic chorea, and in which the lesion was situated in the convolutions. It is a very interesting feature in this case that the choreic movements were preceded by epileptiform crises, which ceased when the choreic movements developed. The choreic movements themselves also disappeared before death. In two

cases of cerebral syphilis, with presumably cortical lesion, I have seen a violent choreiform spasm of the face replace epileptiform convulsions, and there is a close analogy between post-hemiplegic chorea and Jacksonian epilepsy. Dr. Demange reports a case in which hemiplegia was associated with severe tremblings, like those of paralysis agitans, and the lesion was situated in the lenticular nucleus. This form of tremor might, however, be considered distinct from true post-hemiplegic chorea, but in the *Bulletin of the Anatomical Society of Paris*, 1879 (vol. liv. p. 748), is recorded a case in which a true post-paralytic chorea was found to depend upon a softening of the brain on the level of the first convolution, involving the whole thickness of the external capsule, as the sole lesion.

Dr. F. Greiff (*Arch. für Psychiatrie*, 1883, xiv. 598) reports a case where the only lesions were in the cerebral cortex and in the pons; further, that local chorea may be spinal is shown by the case detailed by Eisenlohr (quoted by Foucherand, p. 58), in which choreic movements had existed in both arms from birth, and yet careful microscopical examination failed to detect anything abnormal in the brain, but revealed sclerotic nodules in the cervical cord.

The evidence seems to me sufficient to show that a lesion anywhere in the pyramidal tract—*i.e.*, in the direct line from the brain-cortex to the motor spinal cells—or in the motor spinal cells may produce a localized chorea. When, however, a post-paralytic chorea is associated with hemianæsthesia, the lesion is probably at the position designated by Charcot. The cases reported by Demange separate themselves from those of Charcot in the absence of sensory disturbance.

Chorea of Stumps.—A form of local chorea to which the name Chorea of Stumps was given by Dr. S. W. Mitchell consists in its mildest form of a condition of unstable equilibrium in the muscles of a surgical stump, so that under the stimulus of volition, emotions, or even changes of the weather, they will contract irregularly and spasmodically. In the next degree of intensity spontaneous twitchings and movements occur without any perceptible immediate cause. In severe cases the violence of the movements is such that the stump is perpetually rotated, jerked, vibrated, whirled in all directions, thrashed about, etc. The

spasms in some cases are entirely irregular, in other instances they come and go with clock-like monotony. Appearing first in the peripheral muscles of the stump, they are liable to spread until they involve the whole limb, or even, as in a case which I reported in detail in the *Philadelphia Medical Times*, vol. x. p. 53, one side of the body. In this case the clonic spasms of the flexors of a leg-stump were at the rate of a little over a hundred a minute, each drawing the end of the stump towards the thigh over an arc of from two to four inches. Occasionally there were also spasms of the extensors, and more rarely choreic spasms of the glutei and other muscles moving the thigh. In the forearm the choreic movements occurred from eighty to ninety times a minute. The biceps muscle of the upper arm every now and then was seized with spasms, which for a time were rapidly repeated. The muscles of the shoulders were rarely affected, but the patient stated that sometimes they, with the lateral muscles of the trunk, were very active. There were also slight choreic twitchings of the neck-muscles, and occasionally very decided clonic spasms of the face. The right side of the body was always quiet. Usually in the chorea of stumps the movements cease during sleep; but in the case just spoken of the stump was never quiet, and the patient always, when attempting to go to sleep, placed the arm under his head, so as to hold it still. In most cases, when stump chorea has once set in, it continues indefinitely; usually it is not associated with pain or tenderness. In my own case very careful investigation was made of the nerve-trunks, both by pressure and by the electrical current, without eliciting any abnormal sensitiveness. Occasionally a neuralgic stump is also choreic. The pathology of chorea of stumps remains uncertain. After amputations the nerves are prone to undergo inflammatory changes, which may gradually creep up until they involve the spinal cord. The absence of local tenderness in many choreic stumps indicates that the movements are not due to neuritis; but the only recorded case I know of in which, by section of the nerve, light was thrown upon the question whether the spasm is or is not due to a peripheral irritation is one reported by Dr. Langsdorf (quoted by Mitchell), in which the chorea coexisted with evident neuritis, as was shown by pain and tenderness, and was cured by re-amputation.

Chorea in Internal Inflammations.—There have been reported from time to time cases in which violent, brusque, widespread muscular contractions have been developed in the course of acute internal inflammation, such as a pleurisy, or a bronchitis, or a pneumonia. It is possible that the choreic movements in some of these cases were reflex. The *paramyoclonus multiplex* of Prof. Friedreich, of Heidelberg, appears to be of this character.

Habit Choreas.—There remains a series of local choreas in which no definite cause can be assigned for the spasmodic movements; which movements, also, in a great many cases, closely simulate purposive acts. It is probable that in many of these cases the movements originated during childhood in a frequently repeated purposive act, which soon gained the force of a bad habit, and, not being corrected by the will of the child, grew, in a neurotic temperament, into a fixed custom of the nervous system: hence the term Habit Chorea of Dr. S. Weir Mitchell. A brow may be lifted at intervals, a shoulder shrugged, an eye winked, a jaw dragged forward, a trick of gesture incessantly repeated, even a cough or a snuffle perpetually indulged in. In the beginning these habit choreas are not purely voluntary movements, although controllable by a strong effort of the will, but are allied to the ordinary St. Vitus's dance of childhood, and are greatly benefited by hygienic treatment and by the use of arsenic, as well as by moral means. They finally become so fixed that they are entirely beyond the control of the patient or of any medicinal treatment. Under these circumstances it would appear as though the affected nerve-centres had acquired the habit of discharging themselves at regular intervals independently of any control of the will. The habit chorea has a distinct tendency not only to become more and more uncontrollable with years, but also to increase in its range.

CONTRACTURES.

Contractures may affect one extremity, one lateral half of the body, or the lower extremities, when they are spoken of respectively as monoplegic, hemiplegic, and paraplegic. They may also be confined to a group of associated muscles, or to a single nerve-distribution, or they may exist in scattered unconnected groups of muscles: in a word, as contractures are frequently late

conditions of paralyzed muscle, they follow paralysis in their distribution.

The existence of a contracture is so apparent that it is recognized at once; but much diagnostic skill is sometimes required to determine the cause of it.

Contractures may be clinically divided into those which occur independently of movement,—class A,—and those which take place only during voluntary movement,—class B.

Contractures of class A may be due to—

1. Cerebral affections.
2. Complete loss of power in one of two sets of antagonistic muscles.
3. Chronic neuritis.
4. Irritation of the motor nerve-roots by organic disease of the spinal membranes or of the vertebræ.
5. Sclerosis of the spinal cord, especially affecting the lateral columns.
6. Hysteria.

Contractures of class B are represented by one affection, Thomsen's disease.

Contractures, Class A.

Cerebral Contractures.—Contractures from cerebral hemorrhage may be clinically divided into three sets:

First. Those which come on at the time of the hemorrhage, and which may be known as *immediate rigidity.*

Second. Those which appear from fifteen to thirty days after the hemorrhage, and which may be known as *early rigidity.*

Third. Those which develop after the lapse of some months, and constitute *late rigidity.*

In each of these forms the contracture follows the position of the paralysis, except that the face is rarely implicated, and that the arm is usually more affected than the leg.

Both immediate and early rigidity are the results of irritations of fibres of the pyramidal tract. In their lightest form they may be easily overlooked, but they are to be recognized by the sense of resistance experienced when passive motions of the affected parts are made. They are always associated with an excessive faradic and reflex excitability of the affected muscle, and usually disappear during sleep. As they are the indication of inflam-

matory changes occurring somewhere in the pyramidal tract, they are of serious import.

Late rigidity is due to a descending degeneration of the fibres of the pyramidal tract, and corresponds in its manifestations with the contractures of lateral sclerosis, from which it is to be distinguished by its history, and by its being hemiplegic or monoplegic. The reflexes are always exaggerated. There is finally a progressive atrophy of the muscles, which in the course of years may almost entirely disappear. As has already been stated in the article on Paralysis, the so-called spastic palsy of childhood is often a form of cerebral hemorrhage with secondary degenerations and consequent late rigidity.

Contractures in Infantile Paralysis.—In acute poliomyelitis the contractures and consequent deformities are very slowly developed. The paralyzed muscles remain limp until they are converted into fibrous cords, the contractures being exclusively in their antagonists,—*i.e.*, in the non-paralyzed muscles. Neurologists differ as to whether the distortions are produced by the shortening of the muscles, or the shortening of the muscles by the distortions. Some believe that the non-paralyzed muscles meeting with no resistance gradually undergo alteration and contraction, whilst others believe that the shortening and contraction of the muscles are the result of the settling of the limb towards the origin of the muscle, which causes the muscle to shorten itself for purposes of adaptation. The practical point is, that the non-paralyzed muscles gradually atrophy and grow shorter.

Meningeal Rigidity.—One of the most characteristic symptoms of basal cerebral meningitis is stiffness of the muscles of the neck, due to spasm of the muscles, which, in extreme cases, may cause marked retraction of the head. In the mildest cases there is merely immovability of the head, and even this may be wanting; but when the head is raised from the pillow by the hand, a marked sense of resistance will be felt. This form of tonic spasm is not, strictly speaking, a contracture, but in chronic spinal meningitis the persistent rigidity of the muscles, especially of the legs, may well be mistaken for an organic contracture. The limbs under these circumstances are drawn up on the body, the legs are bent upon the thighs, and the feet are somewhat extended. Relaxation does not occur during sleep or anæsthesia.

Some little difficulty may be experienced in diagnosing between the rigidity of *chronic spinal meningitis* and other organic or hysterical contractures. The symptoms are due to inflammation propagated from the spinal membranes along the nerve-sheaths, so that the spasms are extraordinarily intense, and are associated with violent pains, caused by irritation of the posterior nerve-roots. In rare cases, when the disease is located about the cauda equina, the exudation may produce sufficient pressure upon the nerves to cause paralytic symptoms. Under such circumstances an error in diagnosis is especially liable to occur.

In both organic and hysterical contractures pain is produced by an attempt to straighten the legs; but when the spasms are the result of a spinal meningitis, any attempt to overcome them produces an agony which is much greater than that caused in other contractures. In one or two cases I have been enabled to make the correct diagnosis by noticing the existence of an excessively severe girdle pain. In simple myelitis the girdle sensation may be very pronounced, but it does not rise to the point of agony, as may happen when it is the result of a secondary neuritis of the abdominal nerves. I have noticed, in cases which I believed to be chronic spinal meningitis, tenderness over the large nerve-trunks of the legs, probably the result of a descending neuritis. In a doubtful case aid in diagnosis might be obtained from this, as in myelitis the inflammation travels down the nerve-trunks very slowly, if at all.

Localized chronic spinal meningitis not due to a disease of the vertebræ is usually syphilitic. The diagnosis between it and cancerous tumor must be carefully made. (See pages 50 and 61.)

Contractures of Neuritis.—Contractures do not appear to be a marked symptom of chronic neuritis: when they occur their nature is to be recognized by their history, and by the existence of tenderness over the affected nerves. They may exist in isolated groups of muscles, or they may be symmetrically arranged.

Hysterical Contractures.—Permanent contractures may be caused by hysteria. They may affect one or several limbs, and may be monoplegic, hemiplegic, or paraplegic,—the paraplegic form being, on the whole, the most frequent.

The contractures may affect only single groups of muscles, or may attack a series of muscles surrounding the joints, and in this

way an hysterical club-foot, or an hysterically contracted hand, or an hysterically fixed and apparently inflamed joint, may be produced. In the wide-spread general contracture pain is a rare symptom, but in these localized contractures, especially in the neighborhood of joints, it is very frequent.

The general contractures usually develop suddenly, often following a hystero-epileptic or other violent hysterical attack, and may remain for years, to disappear as suddenly as they appeared. In most cases the shortening of the muscles is excessive, and the rigidity absolute, so that the distortion is extreme: thus, in a contracture affecting the lower extremities, the patient usually lies with the legs rigidly extended, the feet inverted, the heels drawn up to the greatest extent possible, and the toes flexed. In the early periods of the contracture the reflexes are distinctly exaggerated, the faradic contractility of the muscles is increased to a greater or less degree, and the nutrition of the part is good. When, however, the contractures remain for a long time, the muscles undergo gradual wasting, and lose gradually their faradic contractility.

Unless a history of sudden occurrence of the contractures can be obtained, the positive diagnosis of the hysterical contracture is often exceedingly difficult, even in the earlier stages of the disease. It has been asserted that the occurrence of ankle-clonus proves the existence of organic disease; but this is not correct.

In many cases an analgesia and an anæsthesia coexist with contractures. Under these circumstances the diagnosis may be aided by the relative positions of the paralysis and anæsthesia: in cerebral organic hemiplegia the contractures and hemianæsthesia are usually on the same side; in hysterical cases the two symptoms may coexist, or may be upon opposite sides of the body. If contractures are associated with a generalized anæsthesia or analgesia, they are hysterical.

The difficulties of diagnosis are well illustrated in a case which was under my care in the Philadelphia Hospital. The woman suffered from pronounced spinal curvature, due to organic vertebral disease, with contractures of the legs and gross ankle-clonus. No other hysterical manifestations were present, and there was no reliable history of the case. The diagnosis was made of organic degeneration of the spinal cord, secondary to an original transverse

myelitis; but after being in the hospital for many months the patient recovered in a few days.

The question of diagnosis is further complicated by the fact that organic contractures may supervene upon the hysterical variety. Charcot reports the case of a woman in whom contractures of all four extremities developed suddenly and continued for ten years, with but few temporary remissions. After the last seizure the contractures remained until death, and at the autopsy symmetrical sclerosis of the lateral columns was found to extend almost the entire length of the cord. In one of my own cases, contractures which had apparently been originally hysterical did not relax during anæsthesia, and were accompanied with much atrophy of the affected muscle. In accordance with the rule laid down by Charcot, that whenever marked *atrophy of the muscles and persistence of the contractures during anæsthesia* are present *organic degeneration* of the spinal cord has probably set in, the diagnosis in my case would be lateral sclerosis following an originally hysterical contracture.

To sum up, hysterical contractures are to be distinguished from their organic prototypes by—

First. Suddenness of development.

Secondly. An hysterical history.

Thirdly. Presence of anæsthesia or other distinct hysterical symptoms.

Fourthly. Absence of wasting or other changes in the muscles.

Fifthly. Sudden remissions of the contractures.

Sixthly. Absence of various symptoms of organic, spinal, or cerebral disease sometimes present in organic contractures.

Seventhly. Disappearance of the contractures during anæsthesia.

Lateral Sclerosis.—A very common form or cause of contractures is sclerosis of the lateral columns of the spinal cord, either focal or continuous. In the great majority of cases the legs are affected solely, giving rise to the so-called *spastic paraplegia.* But sometimes, especially in focal disease of the lateral columns, the sclerosis may be so limited in the upper portions of the cord that the motor symptoms are confined to one or more of the upper extremities. The diagnosis of lateral sclerosis rests upon—

First. Slowness of development.

Secondly. Gradual loss of power, associated with spasm and heightened reflexes.

Thirdly. Absence of girdle sensation, of pain, or of disturbance of sensation; of paralysis of bladder or rectum, of trophic changes, and of disorder of co-ordination.

As lateral sclerosis has already been fully considered (see p. 66), it is only necessary here to state that in some cases violent tremors develop in the leg during walking and other voluntary movements of the feet.

Contractures, Class B.

Thomsen's Disease.—In *Archiv für Psychiatrie*, 1876, vol. vi. p. 762, Dr. Thomsen described in detail a group of symptoms with which some thirty-five of his relatives were afflicted, and which are now believed to be characteristic of a distinct affection commonly known as Thomsen's disease (*myotonia congenita*, Strumpel). Cases of similar character were described by Charles Bell as early as 1830, and more recently by Benedict in 1868, by Leyden in 1874, and especially by Seeligmüller in the last-named year.* The essential symptom of the affection is that when voluntary movement is attempted the muscle is thrown into a condition of tonic spasm, which may spread to the entire voluntary muscular system, and last for several minutes, before there is sufficient relaxation for the patient to command his actions. In some of the cases the symptoms have dated from infancy, in others they appear to have developed in late childhood or early manhood. Almost invariably the subjects are healthy men, who are apparently extremely muscular, but who, on trial, possess very little endurance, and also comparatively limited muscular power for momentary exertion. The affection is markedly hereditary, and in most of the reported cases clear evidences could be obtained

* The literature of this subject up to the fall of 1883 was very thoroughly collected by Dr. Mobius (*Schmidt's Jahrb.*, cxcviii. 236). Since this time the most important papers known to me are to be found as follows: *Union Méd.*, 1883, xxxvi. 905; 1885, xxxix. 50; *Rev. de Méd.*, 1883, iii. 1064; *Gaz. Hebd. de Médecine*, 1884, xxi. 18; *Brain*, vii. 105–131; *Canada Lancet*, Toronto, 1884–85, xvii. 71; *Alienist and Neurologist*, v. 619; *Centralblatt für Nervenheilkunde*, 1885, viii. 122, 193; *Berliner Klin. Wochenschrift*, 1883, xxii. 605.

of near relatives of the patient having suffered from similar symptoms. In Thomsen's family there was a distinct history of attacks through five generations. Several of his own children were affected, and of his thirteen brothers and sisters seven had developed very decided symptoms. In a remarkable proportion of the cases the disease has been detected in military recruits, who, at first believed to be excessively awkward at drill, were finally found to be really unable to control their muscular movements. Such was the nature of the case reported by Seeligmüller. Peters tells of a soldier twenty years old who, when commanded to march, would remain immovable for a length of time as if rooted to the ground, then, with awkward struggling movements of his arms and legs, he would free himself, and after staggering a few paces would be able to go on regularly; but, even after he had regained control of his muscles, if he attempted to run he would directly fall to the ground in a condition of rigidity which involved his whole muscular system, including the tongue and face. When he laid hold of an object he was unable to let it go; and thus it was with almost any muscular act. In a case which came under my care in this city, the patient complained chiefly that, when going up-stairs, after three or four steps his advancing leg, as he raised himself on it, would be seized with a painless but irresistible tetanic spasm, which would for several moments entirely prevent progression. The tendency to tonic contractures is invariably increased by fatigue, often by emotional excitement, and usually by exposure to cold; warmth, the frequent repetition of movements, and moderate exercise tend to lessen it. Pressure upon the arteries or nerves does not, as in tetany, produce muscular contractions. (Marie, *Rev. de Méd.*, 1883, iii. 1069.)

The contractions may be confined to a single group of muscles in direct relation with those which are primarily caused to contract by the will. Thus, in a case reported by Mobius, the muscles of the leg were the only ones that ever suffered from cramp, which first appeared after excessive fatigue. The case also differed from the ordinary one in the lack of the enlargement of the muscles. Nevertheless, the fact that the patient's father had also suffered marks the disease as the hereditary affection. Dr. L. Deligny (*Union Méd.*, 1885, vol. xxxix. p. 50) details a case in which a brusque turning of the head would give rise to

a spasm of the acting sterno-mastoid muscle. Mastication would produce stiffness in the jaw-muscles. In some cases of Thomsen's disease a sudden push during walking is sufficient to bring on absolute universal stiffness; in others, sneezing, coughing, swallowing, crying, even winking, will produce a paroxysm. A contraction which involves the whole body is affirmed to commence usually in the legs themselves, and to spread to the arms, and finally to the muscles of the hip. This succession probably depends upon the fact that the attacks are generally precipitated by the movements of walking.

Vizioli studied the muscular contraction with the dynamograph (*Alienist and Neurologist*, vol. v. p. 621), and portrayed its peculiarities in graphic curves. "He observed that by making the patient hold the hand open, and ordering him to close it, there passed some five seconds before the movement was accomplished; if the hand was partially closed instead of being widely open,—*i.e.*, if it was put into the state of flexion,—the patient on being told to close it fully did so almost immediately, without the volition being sensibly retarded. This contraction was, however, marked by a line composed of many ample oscillations (*tremor oscillatorius*). The patient took up from five or six to ten seconds in opening the hand, when it had been contracted, and the relaxing of the flexor muscles was not marked in the graphic curve by an almost right line as in the normal state, but by an oblique line with irregular oscillations."

In some cases, as in the soldier studied by Petrone, after the first contractures have passed over, there is complete liberty of action.

In a great majority of the reported cases the contractures have been painless, or, at least, accompanied with no greater disturbance of feeling than the sensation of electric currents running through the part, a sense of swelling, of pricking, etc.; but in the case already referred to as reported by M. Deligny, four or five times a year the patient would suffer a sort of "crisis," composed of a series of general muscular contractions, which, during the period of from twelve to twenty-four hours, recurred at short intervals, repeating themselves upon the slightest provocation, and were always accompanied with such violent pain that the patient would roll on the earth in his agony.

The knee-jerk and the true reflexes are usually normal in Thomsen's disease, but in some cases a direct blow upon the muscle produces a persistent tonic contraction, and Vizioli, and also Marie (*Rev. de Méd.*, 1883, vol. iii. p. 1069), have noted increased knee-jerk. Passive movements are at least in some cases performed without resistance (*Arch. de Neurol.*, January, 1883). To inspection the muscles almost invariably appeared to be hypertrophied, but careful examinations made by Jacusiel, Ponfick, and Petrone have demonstrated that their structure is normal. In a single case Peters found evidences of atrophy in the lower portion of the deltoid. The integrity of the muscular structure is further shown by their electro-contractility being normal, save that momentary application of the faradic current produces a tetanic contraction which may last five and one-half seconds (Seeligmüller) after the removal of the current. In one case Bigouroux believed that the tetanic contraction of closure was greater at the anode than at the cathode. The muscles of involuntary life do not appear to suffer, although in a case reported by Ballet and Marie the larynx was often the seat of the spasm. The pathology of Thomsen's disease is unknown. No autopsies have been reported.

AUTOMATIC MOVEMENTS.

Automatic Movements are complicated movements, closely resembling the purposive actions of ordinary life, which take place independently of the will of the patient.

Cases of automatic movements are best studied for clinical purposes under the headings of—first, those in which the actions are produced by an impulse arising spontaneously within a person, but independently of the will of such person; secondly, those in which the movements occur in response to impulses received from without the person. Cases of the first class are instances of chorea major. Cases of the second class are psychical.

Chorea Major.—In chorea major, or chorea Germanorum, the outbreak is usually preceded by prodromes, such as melancholy, apathy, feeling of nausea, malaise, cramps, or tonic convulsions, disturbances of the circulation, palpitations, etc. The paroxysms usually come on with a general excitement, which perhaps ought

to be considered as a form of aura. During the paroxysms the affected person dances, sings, springs from the ground, rolls himself from side to side, hammers violently with the hands, stamps with the feet, or in a fury of motor excitement whirls with mad rapidity until, completely exhausted, he falls to the ground. The excitement is not confined to the motor sphere: songs are sung, affairs recited, foreign tongues spoken, in a manner entirely beyond the normal power of the individual; events, languages, poetical quotations, which seemingly never have been engraved upon the memory, are recounted or recited in eloquent or incoherent ravings. In the height of the attack consciousness is usually lost, but sometimes it is in a measure preserved, especially in the sporadic cases. As an instance of the sporadic variety may be mentioned a case reported by Robert Watt, in which a girl ten years old turned herself round and round in paroxysms; later, she had attacks in which she would roll from end to end of the bed violently backward and forward, then, lying upon her back, her feet and head would be forcibly jerked together ten or twelve times a minute. A single paroxysm of these movements often lasted fourteen hours a day. In a more recent case, reported by Dr. Bdowenzel (*Schmidt's Jahrb.*, 1874, clxii. 193), a young boy, having warning of an attack, would run home from school, quickly throw himself upon the bed, spring up and down suddenly innumerable times, stand upon his head, cry out, jump from the bed, and run as though in terror round in a circle to the spot from which he had started, not rarely in his fury striking his head severely against obstacles, and performing many other movements, until exhausted, when he would sink upon the bed in a deep sleep, to awake with full consciousness.

Mention has already been made of the religious epidemics of the Middle Ages, which have been in modern times repeated in the outbreaks that have occurred in camp-meetings in the United States (especially in Kentucky in the early part of this century). Of somewhat the same character are the performances of the howling dervishes. The relation of these attacks to hysteria is a very clear one. Undoubtedly, in many cases the paroxysm is brought on by an effort of the will, precisely as the hysterical paroxysm may be induced; but without the hysterical excitement the individual would be incapable of performing many

of the acts which he does. Further, in some cases of chorea major the attacks are really epileptic, being comparable to so-called running epilepsy. (See p. 107.) There appears, however, to be a remnant of cases which can scarcely be considered hysterical, and which certainly are not epileptic. Seemingly of this character are the so-called salaam convulsions of children, in which the paroxysms recur several times a day, last from a few seconds to some minutes, and consist of a bowing forward of the head and body perhaps as many as two hundred times.

Psychical Automatism.—In that form of automatism in which the movements are in obedience to impulses from without, the abnormal condition is a psychical disturbance, which is to some extent illustrated in the phenomena of artificially-induced hypnotism,—a mental condition into the discussion of which I shall not here enter. In some rare cases of insanity the sufferer will do at once that which either by example or by word of mouth he is bidden to do, and will remain almost indefinitely in any position in which he is placed or which he assumes at the word of command. This condition may be mistaken for catalepsy, but is to be distinguished from it by the fact that consciousness is not lost, and that the assumed position is at once departed from when a sharp, quick command is given. I have seen this state of pliability under command extraordinarily pronounced in a scrofulous child of feeble physical organization, but not insane, and of fair mental development.

Miryachit—Latah—Jumpers.—A very curious affection or nervous condition, which is perhaps best classed among the psychical automatic affections, has been noted under various names as occurring in Asia, Europe, and America. The essential feature of this condition seems to be an extreme excitability of the patient, which causes him, upon the least abrupt excitation, such as would be produced by slapping him on the shoulder, hallooing at him, slamming a door, etc., to jump or perform other violent disorderly acts, conjoined with a condition of the cerebral nervous system which necessitates a repetition of voices or sounds (*echolalgia*), or the ejaculation of some word, usually obscene (*coprolalgia*). In some cases the impulse of imitation is so great as to force the victim to repeat not only the spoken word but also any act done by a by-stander. Very frequently the sudden nervous excitement is

accompanied by an excessive emotion, especially of fear, although such emotion may be entirely foreign to the ordinary nature of the individual. The disease appears to be hereditary. It often affects various members of several generations of one family. According to the elaborate description of M. O'Brien (*Journal of the Straits Branch of the Royal Asiatic Society*, Singapore, June, 1883), in Southern Asia the affection is known by the Malay name of *latah*. Mr. O'Brien makes four classes of cases:

Class first, comprising those individuals in whom an unexpected noise produces great alarm, with an irresistible impulse to rush upon the nearest object, and at the same time forces an exclamation which is always obscene.

Class second, comprising those persons in whom certain words when suddenly pronounced will produce an excessive paroxysm of sudden terror. Thus, in an individual noted for his courage and who faced the living alligator without a sign of fear, the sudden pronouncing of the word "buaya" (Malay for "alligator") produced a paroxysm of overpowering terror.

In *class three*, the individuals imitate the words, gestures, or sayings of those in their neighborhood.

In the *fourth class* the individuals become completely abandoned to the will of some other person, performing every act, however *outré* or improper, which they are commanded to do by such individual, standing on their heads, attacking a spectator, etc. In these cases the person who suffers from latah recognizes his enslavement and is greatly depressed thereby, but is unable to prevent it.

According to the observations of American naval officers (*Observations upon the Corean Coast, United States Naval Department*, Washington, 1883), an affection allied to latah exists in Eastern Siberia, where it is known by the Russian name of *miryachit*. In a case seen by the American officers, a pilot would imitate against his will with absolute exactitude all the strange gestures and acts which were performed in his presence by accident or for the determinate purpose of tormenting him, and, even when to escape his persecutors the man had locked himself up in the pilot-house, he could be heard stamping, pounding on the sides of the wall, etc., in exact repetition of acts performed in his hearing by persons without. This case plainly represents class three of O'Brien.

The "Jumpers," or "Jumping Frenchmen," of Maine, described by Dr. G. M. Beard (*Journal of Nervous and Mental Diseases*, vol. vii., 1880), seem also to belong in the present category. In these persons the hearing of a sudden voice or noise causes a repetition of the words or sounds, with the performance of strange antics, whilst a loud command seems to be always obeyed. Thus, a "jumper" was told to throw a knife which he held in his hand: this he did instantly, repeating at the same time the order with a cry of alarm, not unlike that of hysteria or epilepsy. Two "jumpers" standing near each other when commanded to strike each other did so with zeal. Dr. Beard tested the echo-speaking, or repetition, by reading portions of Latin and Greek, when the untutored "jumper" repeated the sounds of the words as they came to him, in a quick, sharp voice, at the same time jumping or making some bizarre motion. The slamming of a door, the falling of a window-sash, or the sudden scream of a steam-whistle produced the same effect as the human voice. In an elaborate review M. Gilles de la Tourette (*Arch. de Neurol.*, vol. viii., 1884, and vol. ix., 1885) has collected a number of cases occurring in an isolated manner in Europe more or less closely conforming to the type of the affection just described.

CHAPTER III.

REFLEXES.

Under the general heading of reflexes I propose to discuss certain movements of portions of the body which are directly produced by external irritations. Some of these movements are probably not of the nature of reflex acts, but the term reflexes is for our present purpose convenient, and is used with the understanding that its employment does not indicate the correctness of any theory as to the way in which the movements are produced.

The reflexes naturally divide themselves into two varieties, the superficial and the deep,—*i.e.*, those movements which are produced by irritations of the skin, and those which are the result of irritation of deeper tissues. This division is not only suitable for the purpose of the clinician, but appears also to be a natural one, as it will be shown hereafter that, whilst the superficial reflexes are probably true reflexes, the deep reflexes, so called, are in all probability not reflexes at all.

SUPERFICIAL REFLEXES.

The superficial reflexes are excited by irritations of the skin and mucous membrane, either by tickling, pricking, pinching, or gently scratching the surface, or by means of a dry electric brush. The nature of these reflexes will be discussed when speaking of the so-called deep reflexes. They are to the diagnostician of comparatively minor import, because, unless it be the plantar reflex, none of them are always present in healthy individuals, whilst some of them are so closely simulated by voluntary acts that it may be impossible to decide whether the movement is the result of volition or of a reflex irritation. Thus, on tickling of the sole of the foot, so long as there is voluntary power and sensation a sudden semi-involuntary and yet truly cerebral withdrawing of the foot is almost sure to occur. It may be laid down as a general rule that the absence of a skin-reflex is of uncertain diagnostic import, whilst the presence of the reflex shows the integrity of

the nerve-arc implicated, such nerve-arc being composed of the afferent nerve, a section of the spinal cord, and the efferent nerve.

In enumerating these superficial reflexes I shall follow closely the work of Professor Ross. According to the classification used by him, there are nine of the skin-reflexes :

First. The *Plantar Reflex*, evoked by tickling the sole of the foot, whose presence proves the integrity of the reflex arc involving the lower end of the cord.

Second. The *Gluteal Reflex*, consisting of contractions of the gluteal muscles produced by stimulating the skin of the buttocks, and depending upon the integrity of the arc through the fourth and fifth lumbar nerves.

Third. The *Cremaster Reflex*, causing the drawing up of the testicle when the skin of the inner side of the thigh is stimulated. Its presence establishes the integrity of the first and second pair of lumbar nerves and their spinal centres.

Fourth. The *Abdominal Reflex*, causing contractions of the abdominal muscles, chiefly the rectus, when the skin of the sides of the abdomen is stroked from the ribs downward. It proves the integrity of the arcs from the eighth to the twelfth dorsal nerves.

Fifth. The *Epigastric Reflex*, causing a dimpling of the epigastrium on the stimulation of the same side of the chest in the sixth and fifth intercostal spaces, and sometimes even in the fourth. This probably requires the integrity of the arcs from the fourth to the seventh pair of dorsal nerves.

Sixth. The *Erector-spinal Reflex*, causing contraction of the erector-spinæ muscles when the skin along their edges is stimulated. It demonstrates the integrity of the reflex arcs in the dorsal region of the spinal cord.

Seventh. The *Scapular Reflex*, causing contraction of some or nearly all of the scapular muscles on superficial irritation of the scapular region. It is evidence of the integrity of the arc of the upper two or three dorsal and lower two or three cervical nerves.

Eighth. The *Palmar Reflex*, producing contraction of the flexors of the fingers on tickling the palm of the hand, and showing the integrity of the arcs through the cervical enlargement of the cord. This reflex is rarely present in healthy adults.

Ninth. Cranial Reflexes, such as contraction of the palatal muscles by irritation of the fauces, sneezing by irritation of the nasal mucous membrane, cough by irritation of the laryngeal mucous membrane, closing of the eyes by irritation of the conjunctiva, movements of the iris by light.

A complete investigation of these cutaneous reflexes in disease appears to be still a desideratum. Rosenbach affirms that the abdominal reflex, and Jastrowitch that the cremaster reflex, are lessened on the paralyzed side in disease of one cerebral hemisphere. It is, however, a universal rule that the withdrawal of the cerebral influence increases the activity of the reflexes, and if the superficial reflexes are really lessened in cases of cerebral disease it must either be in some indirect manner, or else be due to the fact that the lesion which interrupts the motor pathway really irritates the white matter below, and through the white matter the inhibitory centres in the medulla which control reflex acts.

DEEP REFLEXES.

Deep reflexes consist of muscular contractions which are produced by blows upon such deep-seated tissues as muscular tendons and bones. The most widely known and studied of these myotatic contractions are those which are produced by striking the patellar tendon or the Achilles tendon, the so-called knee-phenomenon and foot-phenomenon of Westphal, the patellar-tendon reflex and the Achilles-tendon reflex of Erb. The name of Erb has been shortened by most writers into patella-reflex, and for the term foot-phenomenon the name ankle-clonus has been substituted. The movements of the ankle differ from those of the knee in that they are repeated several times, so as to give rise to a succession of movements. This variation is well indicated by the employment of the term ankle-clonus, whilst the term knee-jerk is coming into vogue as preferable to patella-reflex, as not being indicative of any theory as to the nature of the movements. It is difficult to understand why we should not have these contractions in any muscle whose tendon can be readily reached by a blow when on a stretch; but in the ordinary healthy individual this form of muscular contraction is not readily demonstrated except in connection with the patellar tendon. Occasionally the

biceps of the arm itself may be thrown into movement. In those diseases, however, in which the activity of these so-called reflexes is exaggerated, contractions are possible in many muscles which are not affected in health: thus, it is not rare under the circumstances mentioned for tapping of the tendons of the forearm to produce contractions with movements of the fingers. The so-called *jaw-* or *chin-jerk*, as noted by Dr. Morris J. Lewis, appears to belong to this category. It is best obtained by allowing the jaw to hang passively, or by gently supporting it with one hand, whilst with the other the blow is struck on the chin with a hammer in a downward direction.

To the tendon-jerks which are not usually demonstrable in normal individuals belongs the ankle-clonus. It seems to be affirmed by Mitchell and Lewis (*Med. News*, February 13 and 20, 1886) that the elbow-, ankle-, and jaw-jerks may be occasionally obtained from normal individuals. I have, however, rarely been able to demonstrate them, and the ready production of these jerks in any individual is a strong indication of the existence of disease.

Knee-Jerk.—Of the various muscular contractions, to the diagnostician the knee-jerk and the ankle-clonus are supreme in importance, and I shall discuss them in detail. The condition of the knee-jerk can be roughly examined by seating the patient upon a chair, with one leg crossed over the other, and then striking the patellar tendon below the patella, when a movement of the foot will take place. In order, however, to study closely the knee-jerk it is necessary to have the leg of the patient bare. The blow may be delivered directly on the tendon or upon a rubber band placed across the tendon. In some delicate cases the use of the finger laid upon the tendon for the reception of the blow may be advantageous, as enabling the diagnostician to judge of the force of the blow. The blow may be delivered with the edge of the hand, with the fingers, or with a small hammer having an elastic steel handle and an india-rubber head. The hammer often used in percussion may be employed, or preferably an oblong narrow head may be given to it. For ordinary diagnostic purposes the hand is all-sufficient, and when exaggerated tendon-reflexes are to be judged of, a very slight blow may be delivered with one finger. In some cases, instead of watching the movement of the foot, one hand may be laid upon the quadriceps

femoris and its contractions felt. Another method which is occasionally useful is to allow the patient to sit in an ordinary position with the sole of the foot squarely upon the ground, and then to judge of the effect of the blow by the movements of the quadriceps muscle as felt by the hand or seen by the eye. Dr. A. Money practises still another plan for developing the knee-jerk, with asserted occasional advantage. The patient being in a sitting position, the centre of the instep is taken in the hand and allowed to rest upon its palm or surface at a convenient angle of flexion, and then the blow is delivered. In all cases it is essential to see that the leg be not too much bent, as severe flexion abolishes the knee-jerk. In 1883 (*Deutsche Archiv für Klin. Med.*, vol. xxxiii.), Dr. E. Jendrassik discovered that if a severe muscular exertion be made at the time of the striking of the patellar tendon the effect of the blow is distinctly exaggerated. This observation gave rise to a very elaborate investigation of this subject by Drs. S. Weir Mitchell and Morris J. Lewis (*Med. News*, February, 1886).

These observers found that the knee-jerk varies in health, and is capable of exhaustion by too much use, but may be increased by habitual, not-too-often-repeated, excitation. They further discovered that all volitional acts increase the knee-jerk of either leg, such reinforcement lasting for an appreciable time after the cessation of volition. If, however, the muscular exercise be sufficiently violent and lasting, the knee-jerk is finally enfeebled. Although the contraction of a muscle may be produced when it is in a condition of relaxation, moderate tension increases the reaction, and violent tension, such as is produced by fully flexing the leg, destroys it. The reinforcement of the knee-jerk by volitional act is the immediate result of the volition itself, and not of the act which the volition calls into being. For Mitchell and Lewis found that when an individual who had lost an arm willed movement in the amputated part, the knee-jerk was reinforced. Pain and other sufficiently powerful sensory impressions, such as are produced by the application of heat or cold to the skin, or intense light to the eyes, increase the knee-jerk. It is probably owing to the pain caused that faradic currents applied to the body, and even galvanic currents, have a stimulating effect upon the knee-jerk, an effect which is extremely pronounced when the wire brush is employed with faradism on the dry skin. Galvanic cur-

rents applied to the head increase the knee-jerk, as also do spinal galvanic currents of sufficient power. Pressure upon the sciatic nerve sufficient to produce numbness of the leg decreases the knee-jerk, as does also profound etherization, whilst inhalation of nitrite of amyl has no effect.

Drs. Mitchell and Morris also found that a sufficiently severe blow upon any part of a muscle will produce contraction, which follows the same laws of reinforcement as does the tendon-reflex.

Nature of the Reflexes.—The knee-jerk and all the other so-called deep reflexes are apparently not reflex movements, but phenomena whose immediate causes lie within the muscle directly implicated. Without discussing the evidence in full, it is sufficient to quote the statement of Dr. Ross, that "there is now pretty general agreement among experimenters that the interval of time between the blow and the contraction is not sufficiently long for a reflex act to take place." The contrary to this appears to be true in regard to the superficial or skin reflexes, as the measurements which have so far been made of the time necessary for their development coincide with the period required for a reflex movement. According to the experiments of Dr. de Watteville, about three times as long a time is required for the development of the contraction of the quadriceps femoris after an irritation of the sole of the foot as for the production of the same contraction after a tap upon the tendon. All our present evidence seems to show that the contraction of a knee-jerk arises in the muscle itself, as the result of the stretching of the tendon which the blow causes. It is, however, necessary for the development of the so-called deep reflex that the muscle be in a certain condition of tone, the term tone being here used to express a degree of muscular contraction and irritability, which appears to be the result largely of impulses received from the spinal cord,—these impulses being provoked by peripheral irritations, and being therefore of the nature of minute reflexes. In accord with this theory, impulses especially arising in the muscle itself, or in its immediate neighborhood, stream up to the spinal cord, and by acting upon the ganglionic cells give rise to a continuous series of impulses, passing down to the muscle and maintaining it in a certain condition of activity. Volitional acts evidently increase these muscular reactions by producing a general excitement of all the motor

centres. The impulse-wave which leaves the brain either puts the whole spinal cord in a condition of momentary excitement, or, what is more probable, gives rise to numerous minute impulses, arising in all parts of the spinal cord and flowing down into all parts of the body. A theory which at first thought commends itself is that the volitional act depresses the cerebral centres which inhibit spinal movement, and consequently increases all reflexes by removal of an inhibitive influence: this, however, would seem to be disproved by the discovery of Mitchell and Lewis that the skin-reflexes are not reinforced by muscular acts or by pains. These skin-reflexes are now believed to be of the nature of true reflexes, and if decreased inhibition occurred during volitional acts they ought to be more strongly reinforced than are the myotatic contractions.

Further, the associated movements of Westphal strongly indicate the truth of the overflow theory, since they are best explained by supposing that in a certain excited state of the spinal ganglia the overflow is sufficient to produce definite movements. The overflow takes place in health, but only when the ganglionic cells are sensitized by disease does it produce pronounced effects. When, by disease, there is an interruption of the connections which pass through the spinal centres from the afferent nerve-endings to the efferent nerve-endings, the tone of the muscle becomes enfeebled and myotatic contractions cannot be evoked. When, on the other hand, there is irritation of the afferent nerve-endings, or of the nerve-fibre tracts in the spinal cord, there is an exaggeration of the tone-impulses, and consequently of the muscular tonicity, with a resultant increase in the activity of the myotatic contractions. It is a matter of practical importance to know whether excitement of the motor nerve-trunks is able to increase the tone of the muscle. Sufficient evidence in regard to this point is at present not forthcoming, and even with regard to the sensory filaments in the nerve-trunk we have not an established knowledge; but my own studies incline me very strongly to the opinion that irritation of the sensory nerve does increase muscle-tone.

Whether, as physiologists, we accept or refuse the explanation of these myotatic contractions which has just been sketched, as practical physicians we must recognize that clinical experience

has proved that the knee-jerk and similar jerks are tests in diseases of the nerve-tracts whose exact clinical value is well made out.

Constancy of Knee-Jerk.—It is a matter of vital practical importance to determine the constancy of the knee-jerk in normal individuals. Drs. Mitchell and Lewis found that the intensity of the knee-jerk is greatly lessened by excessive fatigue. Dr. W. R. Gowers (*London Lancet*, November 7, 1885) believes that it is never absent in health. This is certainly contrary to the general opinion of observers, for the reaction has been found wanting by Hufschmidt in 5 per cent.; by Eulenberg in 4.20 per cent.; by Berger in 1.56 per cent.; and by Feilkchenfeld in 1.3 per cent. (*Deutsche Med. Wochen.*, June 6, 1884), of eleven hundred and fifty cases examined by him. Dr. Gowers thinks that the apparent absence has been due to an imperfect examination; but Feilkchenfeld's investigations were made with the greatest care, and Mitchell and Lewis in one of their cases were also unable in any way to get the myotatic contraction. At present, therefore, we must consider that the knee-jerk may be absent in normal individuals, although such absence is exceedingly rare.

Diseases which lessen Knee-Jerk.—The knee-jerk is diminished by lesions that diminish or destroy functional activity in the peripheral nerves or their roots; in the posterior region of the spinal cord,—*i.e.*, in the neighborhood of the posterior nerve-roots; in the ganglionic cells of the spinal cord,—*i.e.*, the motor cells,—or in the muscle itself: consequently the knee-jerk is diminished or abolished in locomotor ataxia, or disease of the posterior columns of the spinal cord; in diffused myelitis affecting the posterior regions or the central portions of the spinal cord; in acute central myelitis affecting the gray matter of the cord; in acute poliomyelitis (whether idiopathic in the child or due to metallic poisoning, as it usually is, in the adult), which causes destruction of the motor cells of the cord; in diseases of the motor nerves, such as traumatisms, neuritis, tumors, etc., diphtheritic paralysis, which interfere with the conducting power of the motor or sensory nerves; in pseudo-muscular hypertrophy with destruction of the muscular tissue; and probably also in fatty or granular degeneration of the muscles. In the first stages of some of the diseases which have been enumerated as destroying knee-jerk there is a condition of

excitation of the tissue which is finally to lose its power, and consequently a condition of exaggerated knee-jerk. This is notably the case in neuritis and myelitis.

There are certain diseases which *a priori* might be expected to destroy the patellar-tendon reaction, but which do not do so. The most important of these is chronic poliomyelitis, or progressive muscular atrophy, as habitually seen in the adult. The explanation of the preservation of the knee-jerk in this affection is not, however, difficult. The individual cells of the ganglionic spinal groups are attacked one by one, and, although a muscle may have greatly wasted, those of its fibres which remain unaffected are still under the normal influence of spinal cells which have so far escaped the disease. In some cases of chronic poliomyelitis a condition of excitation precedes the destruction of the cells, as is especially revealed by the very pronounced fibrillary contractions of the wasting muscles. Under these circumstances the irritability of the muscle may be sufficient to give rise to exaggeration of the tendon-reactions. I have noticed, however, that in such cases the muscles are soon exhausted, so that when the patellar tendon is repeatedly tapped, the reaction, at first excessive, rapidly diminishes in intensity, and at last fails to appear.

How far the tendon-jerks are lost in acute diseases from the loss of muscle-tone which is part of the general degradation, is uncertain. Repeated studies of the condition of the knee-jerk in various chronic disorders not usually attributed to diseases of the nervous system are at present wanting. It would appear probable that when muscular relaxation exists the tendon-reaction would be feeble, but Dr. A. Money (*Lancet,* vol. cclxxxv. p. 842) finds that in all cases of marked typhoid fever, and also of phthisis, the knee-jerk is much exaggerated; and in two cases of rheumatic fever a similar condition existed. In all these diseases the superficial or cutaneous or true reflexes were also grossly exaggerated. It is affirmed that habitually in diabetes the tendon-reaction is lost; but I have seen it exaggerated in that disease. Withdrawal of the inhibitory influence of the brain from the spinal cord is followed by increase of the knee-jerk, and *a priori* it is therefore probable that the knee-jerk may be diminished by rare lesions of the brain of such character and situation as to augment its inhibitory reflex functions. I know, however, of no

clinical proof that stimulation of the motor cortex of the brain is capable of lowering the knee-jerk, unless it be the fact that in a large proportion of cases of general paralysis of the insane (twenty-three out of sixty-five cases: Dr. W. Crump Beatley, *Brain*, April, 1885) it is diminished or abolished. The close connection between locomotor ataxia and general paralysis leads to the suspicion that the loss of knee-jerk is due to posterior spinal sclerosis, a view which is confirmed by Dr. Beatley's report of three cases, in which absence of the knee-jerk during life, without other disturbing evidences of implication of the spinal cord, was found after death to have been dependent upon sclerosis of the posterior columns of the cord. Further, in my own experience, cases of general paralysis with loss of the knee-jerk have habitually suffered from severe pains in the legs, evidently ataxic in character. Again, Dr. Beatley found in two cases of general paralysis in which exaggerated knee-jerk had existed during life pronounced lateral sclerosis of the cord. It appears that in general paralysis there is a very decided tendency to spinal sclerosis, and that the knee-jerk may be absent, exaggerated, or normal, according to the region of the spinal cord which is attacked by the secondary sclerosis.

In diphtheritic paralysis the knee-jerk is diminished or lost, and, as was pointed out by Bernhardt, this loss may precede the paralysis of the palate. It is therefore important in all cases of diphtheria to examine the condition of the knee-jerk during the stage of convalescence. In some cases severe diphtheritic paralysis follows attacks which have originally been so light that their true nature has been overlooked. Under these circumstances the early loss of the knee-jerk is of great diagnostic importance.

In the early stages of pseudo-hypertrophic paralysis the knee-jerk is present, but as the degeneration progresses it becomes less and less, and finally disappears entirely. Occasionally there is some difficulty in diagnosing between a pseudo-hypertrophic paralysis and a very mild spastic palsy of childhood. In the latter disease, however, the tendon-reactions are exaggerated.

It is commonly stated that the knee-jerk is lost in hysterical paraplegia. Dr. W. R. Gowers affirms that this is always an error of observation, due to the inability of the patients to relax the muscle of the thigh.

Of all the diseases in which the knee-jerk may be wanting, it is especially in locomotor ataxia that its absence has diagnostic importance. According to Albrecht Erlenmeyer (*Alienist and Neurologist*, vol. v. p. 455), the loss of the patella-reflex depends upon the sclerosis being localized in the extreme outer portion or external fibres (*bandelottes externes*) of the posterior columns.

In any case of chronic nerve-failure without obvious symptoms or obvious causation, if the knee-jerk be absent there is a probability that the patient is suffering from posterior spinal sclerosis, and if there be conjoined any other symptom of the disease, the diagnosis may be considered as practically certain. Pain is next to loss of knee-jerk in its constancy and diagnostic importance in locomotor ataxia. When unaccountable neuralgic pains occur either singly or in paroxysms in the legs, or pain-crises are present (see chapter on Pain), the condition of the knee-jerk should always be carefully examined. It is remarkable how long posterior sclerosis may exist without producing any loss of co-ordination. A patient of my own, who, until within a few weeks of his death from an intercurrent affection, was an active sportsman, had suffered for fifteen years from almost monthly attacks of furious neuralgic pain in the legs, which had been supposed to be of a rheumatic nature, but which I diagnosed to be due to locomotor ataxia, because the knee-jerk was lost, and because there was no pain on motion, nor soreness of the legs during the paroxysms of suffering. After death pronounced posterior spinal sclerosis was found. The question whether the presence of the knee-jerk proves that the patient has not posterior sclerosis is a very important one. Of all the symptoms of the disease, loss of the knee-jerk is the earliest and most constant, and I should be loath to make a positive diagnosis in any case in which it was preserved. A coexisting lateral sclerosis of the lateral columns might in some measure overcome the depressing effect of a posterior sclerosis. Usually, however, the loss masks entirely the condition of exaggerated excitability; but in some cases the diagnosis may become a matter of difficulty when posterior sclerosis and lateral sclerosis coexist. There must be a stage in commencing posterior sclerosis in which the knee-jerk is only slightly diminished, and it is possible that under such circumstances other symptoms of ataxia may be present in sufficient force to create at

least a suspicion of the true nature of the affection. Dr. Gowers states that he has in the early stage of true tabes seen the knee-jerk present on one side, and has watched its gradual loss, and in one case its gradual return. He further calls attention to the fact that in some rare cases when, as he believes, the true knee-jerk was lost, tapping on the patellar tendon caused a contraction in the extensors of the knee very like that of the true knee-jerk, but which he believes was a true reflex, and not a myotatic contraction, because—

First. On many attempts to obtain the jerk, attempts made under the most satisfactory conditions, no movement could be obtained.

Secondly. The contraction excited was oftener in the flexors of the knee than in the extensors, and frequently it was in the muscles of the opposite leg.

Thirdly. Exactly similar contractions could be produced by a sudden prick of the skin over the tendon of the head of the tibia.

It is not very uncommon for a cutaneous reflex action to persist in early tabes when the myotatic irritability is entirely lost. It is especially in such cases that difficulty of diagnosis arises: thus, the distinction between the local and the reflex contractions is not a matter of mere theoretical interest. In advanced stages of posterior sclerosis, not only are the superficial or skin reflexes abolished, but, if the lesions spread sufficiently high up on the cord, the deep true reflexes may be affected: thus, the power of gargling may be lost. In studying any such case, however, it must be remembered that there are some people who never can gargle.

Diseases which increase Knee-Jerk.—The knee-jerk is increased by brain-lesions which cut off the influence of the cerebral hemispheres from the spinal cord: consequently in most cases of hemiplegia it is increased. In some cases this increase does not appear until eight or ten days after the accident; but usually, if the hemiplegia be at all complete, there is a notable exaggeration in the course of two or three days. Sometimes directly after the apoplexy the knee-jerk is diminished or abolished on the paralyzed side. This is probably due to the propagation of the irritation of the fibres of the brain below the lesion downward to the cerebral inhibitory centres. The increase of the knee-jerk is often pronounced in cases of hemiplegia in which

there is no distinct rigidity; but when either an early or a late rigidity manifests itself, the activity of the myotatic contraction is excessive. Section or lesion of the pyramidal tract in the spinal cord is even more decided in its effect upon the knee-jerk than is a similar organic change situated higher up: hence all affections which interrupt or break the integrity of the spinal cord are accompanied with exaggeration of the tendon-reaction. The most important of these affections are traumatism of the cord, transverse myelitis, and spinal tumors. Again, any lesion which excites, without destroying, the motor ganglionic cells of the cord, increases the knee-jerk: hence its excessive activity in various forms of subacute myelitis. In acute myelitis the lesion progresses so rapidly that the reflexes, at first exaggerated, may be diminished or lost in the course of a few hours or days. In chronic myelitis the knee-jerk is diminished or increased according to the seat and character of the lesion,—*i.e.*, as the organic alteration excites or paralyzes the intra-spinal mechanism connected with the patellar reaction.

Of all chronic affections the one which is especially associated with exaggeration of the various myotatic reactions is sclerosis of the lateral columns. In marked cases of this affection not only will the slightest tap upon the patellar tendon produce violent contractions of the quadriceps femoris, but even a blow upon the tibia, or upon the patella itself, will suffice. Not rarely a single blow will produce three or four or even more successive contractions, and in some cases it is possible to induce a knee-clonus.

Hysterical contractures may be confounded with lateral sclerosis; but the myotatic contractions are usually not so pronounced in hysteria as in the organic disease: nevertheless, they may be just as decided. In a case now under my care, which I believe to be chronic multiple neuritis, the knee-jerk is as active as in a case of lateral sclerosis.

Effect of Disease on Ankle-Clonus.—Ankle-clonus occurs only when the myotatic reactions are exceedingly exaggerated. Its most common cause is lateral sclerosis; but it may be due to hysteria or to subacute myelitis.

Effect of Epilepsy.—According to the observations of Westphal and of Gowers, none of the myotatic contractions can be obtained immediately after a very severe epileptic fit, but at the end of

about half a minute the knee-jerk can again be induced, and frequently it becomes excessive, and during the first few minutes after the fit ankle-clonus may be present. In those cases in which the epileptic fit is unilateral and due to an organic brain-disease, the myotatic contractions are, immediately after the convulsion, usually exaggerated upon the side of the convulsion. Occasionally the myotatic contraction which has been produced in the affected muscle artificially becomes the starting-point of a general seizure. After slight attacks of epilepsy the myotatic contractions often remain as normal, and after moderately severe fits there may be immediately increased knee-jerk and ankle-clonus. The true reflexes are usually abolished for a few moments after a severe epileptic fit.

Effect of Hysteria on Myotatic Contractions.—In hysterical convulsions the myotatic contractions are sometimes normal, but are usually in severe cases increased. According to Dr. Paul Richer, in hysterical catalepsy they are abolished.

The increase of the myotatic contractions in the major hysteria is shown by the excessive effect of a slight irritation upon the muscle directly implicated, and by the tendency to propagation of the myotatic contractions. Further, the character of the myotatic contractions is not rarely altered: they are prolonged, almost tetanic, and after a severe blow may amount to a more or less permanent contracture. According to Richer, the propagation of the myotatic contractions frequently occurs from the leg to the arm, but never in an inverse method, so that a single blow upon the patellar tendon may give rise to muscular contractions involving the whole of one side of the body, whereas a blow upon an arm-tendon affects only the muscles of the neighborhood. When this abnormal neuro-muscular excitability is very pronounced, a slight blow, or even a mere pressure upon the muscle itself, will produce contractions. The effect of striking a bone may be very marked.

CHAPTER IV.

DISTURBANCES OF EQUILIBRATION.

Under the head of disturbances of equilibration I propose to consider three more or less allied, but at the same time quite distinct, symptoms: first, disturbance of co-ordination; second, cerebellar titubation; third, vertigo.

DISTURBANCE OF CO-ORDINATION.

It does not seem to me necessary to discuss here in detail the physiology of co-ordination. For the purposes of the clinician it suffices to define it as that function by which the muscles are so controlled in their movements and relaxations as to execute complicated acts under the impulse of the will. Without the power of co-ordination equilibration cannot exist, but co-ordination may be perfect and yet equilibration be deranged. When the power of co-ordination is lost for the legs, equilibration is affected, because it is impossible for the individual to control the movements of those muscles upon which he depends for his upright position and for the power of walking. If, however, the function of co-ordination be lost in the arms alone, the gait remains perfect, although it is no longer possible for the individual to execute delicate movements with the hands.

Loss of co-ordination is usually first manifested in the legs, because in the majority of cases centric disease begins in the lower portion of the spinal cord, and naturally affects the lower extremities; but when a sclerosis commences in the upper portions of the cord the arms may be the first to suffer. Under these circumstances the patient notices that he is losing the power of doing finer actions with the hands, although the grip and the general strength of the arm may be unweakened. Difficulty is perceived in buttoning and unbuttoning clothes, in picking up pins, threading needles, etc. When the fingers are from any cause anæsthetic, it is difficult for the patient to do many of

these smaller acts, and care is sometimes necessary not to mistake the character of such disablement. A rough test of the power of co-ordination in the general movements of the arm is made by causing the patient to extend the arm at full length, with the hand closed, except the forefinger, and then to bring this rapidly to the point of the nose.

When the power of co-ordination is entirely lost in the legs the patient is unable to stand or walk, even with the aid of crutches. When lying in bed, however, he can kick in every direction, and can execute all movements of the leg with great force. Before this condition of complete disablement is reached there is usually a stage in which the patient is able to walk by means of crutches. Under these circumstances the peculiar erratic method in which the legs are thrown in stepping, the way in which they seem to thrust themselves about, independently of the will of the patient, is characteristic of disordered co-ordination. Preceding the crutch period there is generally a prolonged stage during which the ataxia manifests itself in a peculiar gait. At this time, holding the hand of a second person, or using a cane, is of great assistance in walking. The feet are kept widely apart and straddling, and it is impossible for the patient to walk, or even to stand, with his eyes shut. Very frequently the subject will himself notice that his difficulty of walking is greatly increased at night.

When the loss of co-ordination is very slight, some little care and examination are necessary to detect it. Under these circumstances it will be found that the patient, when his eyes are shut, sways more than he ought to during standing, and also walks with some difficulty. In the very slightest perceptible loss of co-ordinating power the only discoverable derangement may be an inability to stand upon one foot with the eyes closed. In its incipiency the sclerosis of locomotor ataxia is often more pronounced in one side of the spinal cord than in the other: hence a patient may be able to co-ordinate sufficiently to stand firmly upon the one foot, even with the eyes closed, and yet be unable to maintain his position upon the other foot. In a doubtful case the patient should be required to walk backward and to attempt to turn suddenly. Any marked awkwardness in these actions should give rise to suspicion. It is necessary, however, not to confound the

awkwardness arising from muscular weakness, or especially from muscular stiffness due to incipient spasmodic tabes, with that produced by a slight loss of co-ordinating power. Again, in certain cases of cerebral disease with vertigo the patient will execute these movements with difficulty and awkwardness, although the true co-ordinating power is not affected. In some of my patients the first perception of disablement has been in walking through woods or over rough, uneven ground.

Causes of Loss of Co-ordination.

Loss of co-ordination without loss of actual motor power is in the great majority of cases due to sclerosis of the posterior root-zones of the spinal cord,—*i.e.*, locomotor ataxia. It may, however, be a very early or even a prodromic symptom of general paralysis of the insane, and may occur in multiple neuritis.

Locomotor Ataxia.—Loss of co-ordination in the legs without loss of power is so characteristic of locomotor ataxia that the gait it causes is commonly known as the *ataxic gait*. When a posterior sclerosis is sufficiently advanced to affect progression, but has not yet reached the stage in which a stick or other support is necessary, the patient walks with his head a little bent forward and the eyes directed to the ground. The trunk inclines upon the thighs, whilst the feet are held in advance of the buttocks, with the legs widely separated from each other. At the same time, owing to the excessive contractions of all the muscles of the lower extremities, the leg proper is extended somewhat rigidly upon the thigh, and there is very little movement at the knee-joint. The advancing leg is therefore raised from the ground in some degree by an elevation of the pelvis, although at the same time some flexion does occur at the knee-joint. By these conjoint movements the foot is freed from the ground, and, having been flung forward and outward by a rapid muscular jerk, comes down with a thump like a solid mass. In some cases the heel is the last to leave the ground and the first to touch it. Not rarely the pelvis is so much inclined during walking as to carry the centre of gravity too far towards the side of the stationary leg. To counteract this and maintain the balance of the body, the upper portion of the trunk is curved towards the advancing leg by a contraction of the erector-

spinæ muscles, or the arm corresponding to the advancing leg is thrust out laterally. The alternation of these movements at each step may give a pendulum-like swing to the body. In a more advanced stage of locomotor ataxia the patient is able to walk only by the help of two sticks or crutches. The body is thrown forward, in order to counteract the tendency to fall backward produced by the peculiar position assumed by the legs, which are held in advance of the buttock on account of the tendency to undue contraction of their extensor muscles; the foot is usually at an obtuse angle to the leg, and the thigh at an obtuse angle to the trunk. If under these circumstances the trunk be erect, the line of the centre of gravity would fall through the buttocks posterior to the point of support,—*i.e.*, the foot,—and consequently the patient would fall backward. To overcome this, the trunk is often bent so far forward that the line of the centre of gravity is in front of the feet, and the patient would fall forward if he were not supported by a stick or crutches. All the movements executed with the legs are performed with great stiffness and by sudden jerks. The straddle is usually very marked, and the leg is raised from the ground by an elevation of the pelvis in the method already described. Still later in the disorder the legs are entirely beyond the control of the patient. They are thrown about in wild, irregular, choreiform movements, which render them of no use whatever in walking. Under these circumstances progression is impossible. When the lesion travels up the spinal cord all power of co-ordinating the muscles of the trunk may be lost, so that the patient is no longer able to sit in a chair.

General Paralysis.—In general paralysis of the insane, the early loss of co-ordination is felt almost exclusively in the hands, and is shown chiefly in delicate skill-requiring acts, such as writing, engraving, etc., whilst in locomotor ataxia it is extremely rare for the arms to be first attacked. The other symptoms of the two diseases are in no way similar: locomotor ataxia is, however, a very common complication of general paralysis. (See General Paralysis.)

Multiple Neuritis.—Multiple neuritis affecting the sensory nerves is always accompanied not only with pain, but also with pronounced tenderness over the nerve-trunks, which at once distinguishes it from locomotor ataxia. There is, however, evidence

that a multiple neuritis is often incited by, or at least follows upon, chronic posterior sclerosis.

Loss of Co-ordination as a Complicating Symptom.—In multiple sclerosis, and in certain forms of chronic myelitis, the posterior column shares the lesion along with other portions of the cord. Under these circumstances the loss of co-ordination is associated with various symptoms, such as palsy, spasm, etc., due to other portions of the cord being affected, and, indeed, may be so entirely masked by these symptoms that its presence cannot be detected.

TITUBATION.

Cerebellar Affections.—If a lesion be confined to one hemisphere of the cerebellum it may produce no symptoms whatever, and in any case cannot be diagnosed with certainty. Vomiting, with occipital headache and general failure of health, might in some of these cases lead to a suspicion of the seat of the disorder, but these symptoms may be entirely wanting, as is shown by a case reported by Dr. Loomis (*Amer. Med. Times*, 1862, iv. 124), in which the symptoms simply resembled those of a low fever, although a cerebellar tumor the size of a small orange was found after death. When, however, a growth or other lesion of one hemisphere of the cerebellum causes such enlargement as to exert pressure upon neighboring parts, various paralyses result. The encroachment upon the medulla may lead to an imperfect hemiplegia or even to general motor failure, or hypoglossal, facial, or other local paralyses may result from the pressure exerted by the enlarged hemisphere upon nerve-trunks. If the trigeminus nerve be involved, a true anæsthesia dolorosa may be produced: loss of the power of swallowing may also be a prominent symptom. On account of the proximity of the corpora quadrigemina, blindness from pressure is a not infrequent result of cerebellar tumors. When the cerebellar lesion occupies the middle lobe it causes peculiar disturbances of motion, which are pathognomonic, and to which the name of *cerebellar titubation* has been given. Very frequently cerebellar titubation is associated with giddiness, but, as in some instances giddiness is absent, the disorderly movements are plainly not caused by the vertigo.

Gait in Cerebellar Disease.—The position which is assumed by

the victim of cerebellar titubation during standing resembles that of locomotor ataxia. The feet are held well forward and widely separated from each other. If the attempt is made to bring them close together, peculiar movements of extension and flexion occur in the feet, and at the same time the trunk begins to rock and stagger more and more violently, until, in extreme cases, the subject falls unless he can seize some support. In unusual instances the movements are definite and in one direction; but commonly they are irregular, and vary both in direction and in force. The staggering may be so great that the patient is unable to move a step. Very commonly it is impossible for him to turn suddenly without falling. Sometimes the symptoms are intensified by darkness or by closing the eyes, whilst in other cases they are not thus affected. The walk resembles that of an intoxicated man. There is a similar staggering, with to-and-fro movements of the whole body, resulting in a zigzag instead of a straightforward progression. In most cases the feet are raised only a short distance from the ground, and are moved with a peculiar irregularity of step. In some instances the patient has a tendency to fall or run backward, or this may be reversed and the patient continually falls or runs forward. This is, however, by no means a constant phenomenon, nor is it when present absolutely characteristic of cerebellar tumor. At least I have seen cases in which a similar symptom existed when there was no other reason to suppose a cerebellar tumor: in no instance, however, have I been able to confirm the diagnosis by an autopsy. The movements in titubation are sufficiently distinct from those of ataxia to make their recognition in most cases easy. A further difference is to be found in the fact that whilst in cerebellar disease the patient lying in bed is able to move his legs with normal promptness and accuracy, in spinal disease the movements in bed are almost as disorderly as during walking. Further, whilst ataxia often affects the arms, titubation is confined to the lower extremities. It is, indeed, due to disorder of equilibration, and not to any loss of muscular control, and appears only when the attempt is made to exercise the function of equilibration.

Diagnostic Value of Titubation.—Titubation is probably pathognomonic of disease of the cerebellum, and, as Nothnagel has shown, of the middle lobe of the cerebellum. There have,

however, been cases in which the middle lobe of the cerebellum has been involved without the production of titubation. The explanation of Nothnagel, that this has been because sufficient of the middle lobe to perform its function has escaped injury, may be accepted, at present, as at least the best that can be given.

Rotatory Movements.—Titubation must not be confounded with the rotatory movements which occur when the cerebellar peduncles are implicated, either as they enter the pons or higher up. These rotatory movements, the "movements of manége," are around the long axis of the body. Prof. Rosenthal sums the diagnostic points of *tumors of the cerebellar peduncles* as headache, vertigo, disorders of the special senses, hemiplegia, unsteady gait, with a tendency to fall upon the side, and partial rotation around the vertical axis, with lateral rotation of the head. There have, however, been recorded a number of cases of lesions of the cerebellar peduncles without rotatory movements, and it is probable that such movements, when present, are produced in some indirect manner.

VERTIGO.

Vertigo may be defined to be a sensation of moving, or an appearance of motion in surrounding objects which are really at rest. It is a sense of defective equilibrium without actual disturbance of position, and varies in intensity from the slightest giddiness to that condition in which everything about the victim seems to be involved in a whirling chaos of motion. In the slighter forms of the symptoms, those to which the term giddiness is well applied, there is a feeling as though the head itself or its contents were in motion: hence the popular term "swimming in the head." Closely allied to this mild vertigo is the sensation of rising through the air, which almost every one has experienced after fatigue when lying in bed. An abnormal sensation somewhat similar to this, but more distressing and terrifying, is that of falling through the air, which in extreme cases is accompanied by a feeling as though the earth were opening and rising up to swallow its victim. In vertigo proper the movement is in the surrounding objects: the furniture and other contents of an apartment appear to revolve more or less rapidly, to dance backward or forward, or to reel

with an irregular, staggering gait. The ground rises, or sinks, or rises and sinks like the waves of the ocean. Houses move, hills, trees, and rocks slant hither and thither, and in some instances the whole landscape inverts itself and hangs above the head, threatening ruin.

In vertigo relief is generally afforded by assuming a horizontal position, or even by the closure of the eyes, but in severe cases these measures fail, and the patient lies in bed clutching at any available support, in constant fear of falling. In many cases along with the vertigo there are distinct perversions of special senses. Mistiness of vision, enlargement or lessening in the size of objects, tinnitus aurium, the rush of water, intermittent pulsations, the clanking of pumps, the hissing of teakettles,—these and many other extraordinary alterations of perception, or even absolutely subjective sights and sounds, may form a part of the vertiginous paroxysm. In the majority of such cases, however, disturbance of the special senses is the origin of the vertigo, or the subjective sensations and the vertigo depend upon a common cause.

Vertigo may be present almost all the time, or at least be produced by every change of position, or even by the erect posture, or it may come on at irregular intervals and be of a purely paroxysmal type. To the condition in which paroxysms of vertigo succeed one another in rapid succession the name of the *vertiginous status* has been given by Dr. S. Weir Mitchell,—a name which was evidently suggested by the parallel between this condition and the epileptic status. When the type of the disorder is strictly paroxysmal the attacks are often very severe, and are accompanied by nausea and vomiting, and even by relaxation of the bowels and the rapid secretion of a limpid urine, like that of the hysterical fit. The gastro-intestinal disturbance in a large proportion of these cases is secondary to the vertigo, but, as will be discussed in detail later, the vertigo may be dependent upon the gastro-intestinal irritation. In severe vertigo there is frequently some mental confusion, which may end in complete loss of consciousness. When this happens, the vertigo is probably due to hysteria, epilepsy, organic brain-disease, or uræmia. As insisted upon by Dr. Mitchell, a distinct aura sometimes precedes the vertiginous paroxysm, or in some cases there is an abrupt onset with the sensation of a snap in the head; more rarely the vertigo is ushered in by a sensory dis-

charge, such as the perception of light or sound. In such cases there is reason to fear that the vertiginous attack is allied to epilepsy.

Nature of Vertigo.—The theory that vertigo is produced by disturbance of the circulation of the brain has met with widespread acceptance, but I do not think it can be received as a general theory applicable to all cases. I am not prepared to enter into a discussion of the theory of vertigo, but it seems to me probable that at least two, and perhaps more, distinct conditions are habitually united under the one name, because the sensations which accompany them are similar. The vertigo of epilepsy, the vertigo of organic brain-disease, and the so-called laryngeal vertigo are probably caused by nervous discharges allied to those which provoke epileptiform convulsions, whilst the gastric vertigo and many toxæmic vertigoes are of different character. The epileptiform vertiginous attack is often preceded by an aura, and naturally ends in unconsciousness, whilst the typical gastric vertiginous paroxysm has no aura, and terminates in vomiting.

Causes of Vertigo.—The diseases upon which vertigo may depend, or of which it is a symptom, can best be studied under eight headings:

1. Organic Vertigo, in which the symptom is dependent upon some demonstrable structural alterations of the brain or spinal cord.

2. Cardiac Vertigo, in which the vertigo depends upon some evident alteration of the circulation.

3. Epileptic Vertigo, in which the attack replaces a paroxysm of idiopathic epilepsy.

4. Hysterical Vertigo, in which the symptoms are hysterical: in this division I shall include those cases in which the vertigo is the result of nervous exhaustion.

5. Peripheral Vertigo, in which the paroxysm depends upon an irritation of some peripheral nerve-filaments.

6. Vertigo of the Special Senses, which is caused by some derangement of the special senses.

7. Toxæmic Vertigo, in which the symptoms are toxæmic, due to a mineral or a vegetable principle, or to a disease-poison in the blood.

8. Cases in which at present no explanation of the vertigo

is forthcoming, and for which the name of Essential Vertigo has been proposed by J. Spence Ramskill.

Organic Vertigo.—Chronic meningitis, brain-abscesses, specific, cancerous, or simple tumors, atheroma of the basal arteries, and almost any chronic brain-disease producing or accompanied by coarse structural alterations, may be the cause of vertiginous attacks. Vertigo is apt to be especially severe when the focal disease is situated in the cerebellum, but cerebellar atrophy, and even cerebellar tumors, may exist without pronounced giddiness, and a tumor may be located in any portion of the brain, even in the extreme frontal lobes, and yet cause giddiness. Organic vertigo is in the majority of cases not severe, although it has a distinct tendency to end in unconsciousness. I cannot remember a case in which the cerebral hemispheres were alone implicated in which the sense of movement either of the person himself or of surrounding objects was very violent. The recognition of the cause of the vertigo in cases of structural brain-disease is to be based upon the other symptoms of the case.

In general paralysis of the insane vertiginous attacks are not rare. They must be looked upon as an abortive form of the epileptic convulsions which are common to these disorders.

According to Charcot, vertigo marks the invasion of multiple cerebral sclerosis in about three-fourths of the cases. I have seen a large number of cases of this disease, and vertiginous attacks have certainly been the exception. Charcot says that the vertigo is usually gyratory; all objects are apparently whirling round with great rapidity, and the individual himself feels as though revolving on his axis. Charcot further states (*Diseases of the Nervous System*, Phila., 1879, p. 160) that "the vertigo in question is all the more interesting because it belongs neither to locomotor ataxia nor to paralysis agitans, and may consequently help in forming a diagnosis." Notwithstanding this statement, vertigo is a not very rare symptom in locomotor ataxia. This seems to be true not only of cases like those reported by Fournier (*De l'Ataxie locomotrice*, p. 251), in which the disease is really not locomotor ataxia but cerebro-spinal syphilis, but also of genuine posterior spinal sclerosis. In the last-named affection the giddiness occurs especially in those cases which have marked ocular or aural disturbance or severe gastric crises. It is very probable

that in such instances the vertigo is a secondary and not a primary symptom of the disorder,—*i.e.*, is caused by the peripheral irritation or the sensory disturbance. Dr. S. Weir Mitchell, however, affirms that vertigo may occur in locomotor ataxia independently of ocular disturbance, and T. Grainger Stewart (*On Giddiness*, Edinburgh, 1884) insists that the vertigo may be due to the centric lesions.

Epileptic Vertigo.—Attacks of giddiness of the mildest possible type to be noticeable may be a symptom, or rather a paroxysm, of a hopeless idiopathic epilepsy. Frequently the nature of such a paroxysm is mistaken. The epileptic vertigo may be scarcely perceptible, or it may be severe and end in disturbance of consciousness. There is nothing in the vertigo itself upon which the diagnosis of its nature can be made. The judgment must be based upon concomitant circumstances, such as known hereditary tendency to epilepsy, absence of the ordinary known causes of vertigo, age of the patient at which the vertigo appeared, etc. A previous history of convulsions during childhood, with persistence of the vertiginous paroxysms, would be decisive. If in any case recurrent vertigo be ushered in by an aura, and be followed by mental disorder, a sense of transportation through space, a marked subjective sensation, such as that of a bright light, or of a loud sound, suspicion should be strongly roused unless the subject be hysterical. When any disturbance of consciousness, muscular rigidity, or clonic convulsive movements accompany the vertigo, the prognosis becomes grave. Such vertigo, if not hysterical, is almost invariably organic or epileptic. The occurrence sooner or later of a pronounced epileptic paroxysm will generally settle the diagnosis. Dr. George Parker (*Brain*, vii. 525) affirms that in epileptic vertigo there is "always falling towards one side, never, as in brain-disease, a sense of spinning round, nor, as in eccentric vertigo, of the room moving." The correctness of this statement seems to me extremely doubtful.

Cardiac Vertigo.—Vertigo is a not rare symptom of chronic cardiac disease, especially of fatty degeneration, or other diseases of the heart, accompanied by failing power. In some of these cases abrupt alterations of position, especially sudden rising from the bed, or prolonged stooping, may produce a vertiginous paroxysm. Even in the normal individual it is not rare for rapid forced

breathing, prolonged standing with the head downward, violent straining at stool, excessive vomiting, or other acts which cause marked disturbance of the circulation, to provoke giddiness. In cases of doubtful organic brain-disease I have sometimes been aided in making the diagnosis by the ease with which excessive giddiness was produced by acts like those just spoken of. The giddiness which forms a prominent symptom of the *mal de montagne*, an affection caused in some persons by the rarefied air of high mountains, and manifested by headache, vertigo, and dyspnœa, with sometimes nausea and vomiting, is probably due to disturbance of the circulation. The giddiness of anæmia and that of plethora with excessive cardiac action probably have similar explanation. Sudden loss of the cerebro-spinal fluid,—abrupt changes of atmospheric pressure, such as is experienced in going from a chamber containing compressed air into the ordinary atmosphere,—these and other conditions or acts not necessary here to detail may cause giddiness by disturbing the brain-circulation.

Giddiness is very common in anæmia. Indeed, it may be said to be a constant symptom, if only the anæmia be sufficiently pronounced. Severe vertigo is, however, rarely, if indeed ever, caused by anæmia, since in extreme cases the giddiness soon merges into syncope. Anæmic giddiness is prone to be especially developed by changes of posture which suddenly affect the blood-supply, such, for instance, as abruptly rising from the horizontal to the erect posture. After protracted illness, during the feebleness of convalescence the first attempts at getting up are apt to cause swimming in the head.

The vertigo which occasionally develops in persons of advanced age may be considered as an organic vertigo, or as one due to disturbance of the circulation, for it probably depends upon a lack of blood-supply to the brain-cells, the result of the atheromatous degeneration of the vessels. When once developed it is apt to be a persistent, obstinate symptom. A vertigo of similar character may be caused by syphilitic or gouty changes in the cerebral vessels, and occasionally precedes brain-softening.

Hysterical and Neurasthenic Vertigo.—Vertiginous sensations are not a prominent symptom of hysteria, and when present are apt to take some unusual form. Almost any variety of vertigo may, however, be so closely counterfeited by the hysterical

disorder that great care will be necessary to avoid error in diagnosis. This is especially true when tinnitus aurium or other sensory disturbance coexists with the vertigo and affords a picture of organic brain-disease. A diagnosis of such organic disease should be made with great reluctance whenever there is a pronounced hysterical temperament.

In neurasthenia giddiness or swimming in the head is moderately common, though rarely, if ever, severe. It seems sometimes to be connected with lack of proper blood-supply to the brain, and so far to be related to anæmic vertigo; it is also based to a greater or less extent upon a morbid sensitiveness of the nerve-centres, and is provoked by peripheral irritations which in health make no impression. Hence bright lights, as the flashing of a mirror, loud sounds, bad smells, etc., may in a neurasthenic produce a giddiness which is in a sense ocular, aural, or nasal.

Neurasthenic vertigo is often the result of long-continued overwork, of sexual excesses, or of prolonged lactation. Indeed, almost any persistent depressing cause may bring about the bodily condition which produces vertigo. It is evident that this form of vertigo is in many cases allied to anæmic vertigo, since nerve-exhaustion and poverty of blood not rarely coexist.

Peripheral Vertigoes.—Vertigoes which are due to irritation of some peripheral nerve-filaments constitute a numerous and important class, in which are included laryngeal, gastric, and intestinal vertigo.

In 1876 (*Gaz. Méd. de Paris*, 1876, p. 588), Prof. Charcot described, under the name of *laryngeal vertigo*, several cases of an affection that has since been repeatedly observed. The attack begins with a burning or itching in the larynx, that causes in a moment a violent access of spasmodic cough, which is soon followed by a brief vertigo, ending in complete loss of consciousness, lasting for a very few minutes, during which, in some of the cases, there have been convulsive movements of the face and even of the extremities. The paroxysm is not followed by nausea and vomiting, as is ordinary severe vertigo, nor yet by sleep, as is typical epilepsy.*

* See Dr. Gasquet, *Practitioner*, August, 1878; Dr. Sommerbrodt, *Berlin. Klin. Wochenschrift*, September, 1876; Dr. Krishaber, *Annales de l'Oreille*

Within certain limits the symptoms have varied considerably in the recorded cases. Usually the cough is severe, but in some instances it has been slight. The vertigo may be pronounced, but seems to have been in most cases very mild, and in some altogether wanting. Consciousness, often completely lost, may be imperfectly preserved, or may be even unaffected. The occurrence in the same case (that of Dr. Lefferts) of attacks varying from the slightest vertigo to complete unconciousness shows that a unity of character runs through the varying paroxysms. There has been considerable discussion as to whether these attacks should be called vertiginous or epileptic. But the question is probably one of words merely. There is probably no difference, except in intensity, between some forms of vertigo and an epileptic attack. One constantly replaces the other in an idiopathic epilepsy. The probable explanation of laryngeal vertigo is that a reflex nervous discharge is caused by the laryngeal irritation. In some of the cases gross laryngeal lesions (polypus, Dr. Sommerbrodt) have been noted, in others redness of the laryngeal mucous membrane, in others no lesion. Asthma has in one case apparently caused the attack, the onset of which was felt in the trachea. (*Le Progrès Méd.*, 1879, p. 317.) When laryngeal disease has been found, its cure has been followed by relief; and even when no lesion has been apparent, cauterization of the larynx has done good.

I think it is certain that vertiginous and epileptoid attacks may be produced by a peripheral laryngeal irritation; but some care may be necessary not to mistake a true epilepsy commencing with a laryngeal aura. Laryngeal crises of locomotor ataxia also may simulate a laryngeal vertigo. It is probable that in some cases an attack of unconsciousness may, as insisted upon by Dr. Elsberg, be precipitated by a spasm of all the laryngeal abductor muscles, arresting respiration.

Gastric vertigo occurs in an acute form as the result of an acute indigestion or gastric irritation. In some individuals the indulgence in strawberries, lobsters, shell-fish, or other article of diet to most persons harmless, invariably produces a severe vertigo, undoubtedly by irritating the gastric nerves. Irritation of the

et du Larynx, viii.; Dr. George M. Lefferts, *Trans. Amer. Laryngol. Soc.*, 1883; Dr. Charcot, *Le Progrès Médical*, April, 1879.

mucous membrane of the stomach not intense enough to cause a vertiginous paroxysm may produce great flushing of the face or an intense pain just below the ears. Acute indigestion with excessive acidity may provoke an intense reflex headache or a violent attack of vertigo, the paroxysm in either case often being accompanied by partial blindness or double vision, and finally by nausea and vomiting, followed by relief. Not rarely the headache and the vertigo are both present.

Chronic gastric vertigo, due to persistent dyspepsia, is a much rarer affection than was supposed by Trousseau and his followers. In those cases of chronic dyspepsia in which the more or less constant vertigo is at its worst two to four hours before eating, it seems to me as rational to ascribe the vertigo to the presence in the blood of products of imperfect digestion as to attribute it to gastric irritation. In some dyspeptics, however, there are more or less frequent paroxysms of vertigo, with ocular disturbance and sick stomach, closely simulating those of an acute gastric vertigo.

It is possible that the vertigo which occurs long after eating in chronic dyspepsia may sometimes be due to an intestinal irritation, as is undoubtedly the giddiness with a sense of weight over the brows, or even of burning in the eyes, which may be the only manifest symptom of tapeworm.

Vertigo from the Special Senses.—It is well known that certain rapid changes of position produce giddiness, notably rapid whirling, as in the waltz, swinging, as in the play of children, and the rocking motion of the ocean. The peculiar sinking feeling which is experienced in the abdomen during the descent of the swing and during the going down of the ship into the trough of the sea indicates very strongly that the vertigo and the giddiness which accompany these movements are, at least in part, the result of afferent impulses which are produced in the abdominal viscera by the rapid assumption of positions to which they are unaccustomed. On the other hand, the relief which to some extent is secured in all these cases by closure of the eyes indicates that the rapid passage of objects in abnormal positions or abnormal succession before the eyes is at least one factor in the production of the vertiginous sensations.

Ocular Vertigo.—Various ocular defects or diseases cause ver-

tigo. The most frequent of these is paralysis of the external rectus; but any muscular palsy which causes a discord in the optic axis may produce vertigo. Under these circumstances closing the affected eye usually puts a stop to the giddiness. In most cases shutting the sound eye does not produce relief. The vertigo which in these cases is present when both eyes are open is probably the result of the confusion of the nerve-centres produced by the non-agreement of the eyes in their representation of objects. The giddiness which exists after closure of the sound eye is probably due to the discord which still remains between the visual perceptions on the one hand and the sensations arising from the muscular sense and general sensibility on the other. The object is seen in one direction but felt in another, or as directed by the eye the muscles assume a certain position in order to maintain the erect posture, but the common sensibility and the muscular sense enforce the necessity of another posture. In this way a confusion of the lower brain-centres is produced, which results in vertigo. Dr. T. Grainger Stewart has reported (*On Giddiness*, Edinburgh, 1884) a case of nystagmus in which the vertigo was very strongly marked, but was at once overcome by holding the eyeballs forcibly quiet,—a very strong indication that the giddiness which is sometimes present in nystagmus is the result of the rapid changes in the position of sensory impressions on the retina. The reason that so many persons with ocular palsies or with eyes which are not optically in accord do not suffer from giddiness is that the habit is soon acquired of neglecting the images formed in one retina, or, in other words, of using only one eye in conscious vision.

Aural Vertigo.—In 1861 (*Gazette Méd. de Paris*), P. Ménière described a case in which a young man was suddenly seized with a violent vertigo, accompanied by deafness, pallor of the face, excessive sweating, and apparent symptoms of an imminent syncope. He fell to the earth without being able to raise himself up, and, lying upon his back, could not open his eyes without all the surrounding objects seeming to whirl in space. The slightest movement even of the head increased the vertigo and produced violent vomiting. In a second case, a young woman, after exposure during her catamenial period, suddenly became deaf, with violent vertiginous attacks similar to those just described. Five days later she died, and at the autopsy the brain and spinal cord were

found normal, but in the semicircular canals there was a bloody exudation of which scarcely a trace could be perceived in the vestibulum. These and other similar cases led Dr. Ménière to recognize a form of violent vertigo produced by intense congestion or apoplexy in the semicircular canals. Since the publication of the papers of Dr. Ménière, numerous articles have appeared describing vertiginous attacks in connection with diseases of the middle ear. Probably all the diseases of the semicircular canal are liable to be associated with vertigo, but the name Ménière's disease should, I think, be restricted to those cases in which the vertiginous attacks are due to an apoplexy or a sudden congestion. I have seen violent persistent vertigo resembling that of Ménière's disease caused by a small pistol-bullet lodged somewhere in the vicinity of the semicircular canals. The relation between the aural apparatus and the function of equilibration is undoubtedly a close one. As has been shown by Dr. S. Weir Mitchell, the injection of cold water or of rhigolene into the external meatus gives rise to convulsive movements in the rabbit and guinea-pig, with, on repetition, the production of a permanently vertiginous state. In man, cold water suddenly thrown into the ear will sometimes cause excessive vertigo, as in the case of Dr. Mitchell himself, in whom a jet of water at a temperature of 52° F. into the left ear was immediately followed by disturbance of vision, with movements of surrounding objects to the left and a fall to the left. After getting up, there was swimming of the head and a sense of lack of power in the whole left side, with staggering to the left. The relation of such an attack as this to various reflex vertigoes and epilepsies is a very evident one. The closeness of the relation is still further enforced by the fact that in birds many parts of the skin are competent under irritations to give rise to vertiginous phenomena. It does not appear to me that the ease with which vertigo is caused by irritations of the external ear proves that the external ear is in direct connection with the function of equilibration: the phenomena are readily explained as reflex. The cause of the vertiginous attack in labyrinthine disease is as yet uncertain. By most physiologists it is believed at present that the canals have a very direct relation with equilibration, or are, in other words, guiding organs. It is, however, possible that the vertiginous sensations which their injuries produce are purely

of the nature of a reflex disturbance, having no more immediate connection with equilibration than have similar vertiginous attacks produced by permanent laryngeal and gastro-intestinal irritations.*

Toxæmic Vertigo.—Cannabis indica, alcohol, belladonna, and various other poisons are capable of producing a more or less pronounced vertigo. In such cases the cause of the vertigo is to be recognized by the presence of other symptoms of poisoning, and by the history.

As long ago as the days of Boerhaave, the possible dependence of vertiginous symptoms upon irregular gout was recognized. This vertigo of lithæmia may be very mild or very severe. The attacks may occur at long intervals or may be repeated several times in the twenty-four hours. In the severer attacks the whirling of objects is very pronounced, and the confusion of mind may be marked. In some of these cases there is along with the severe vertigo an apparent loss of memory, which is liable to lead to a mistaken diagnosis of organic brain-disease. Almost always irregular or shooting pains, depression of spirits, irritability, malaise, or other evidences of suppressed gout can be noted, and should lead to an examination of the urine, which will reveal the presence in it of uric acid or the urates, and confirm the diagnosis. In some cases gouty vertigo is associated with marked irregularity of

* *Voltolini's Disease.*—A disease which is related to aural vertigo, but presents symptoms more closely resembling those of basal meningitis, was originally described by Dr. Voltolini. It is almost absolutely confined to childhood. The attack is sudden, sometimes preceded, however, for some hours by restlessness, with shooting pains in both ears. Unconsciousness now develops, often with great suddenness, and is associated with high fever, great restlessness, contracted pupils, and strabismus; delirium is occasionally present, and convulsive movements or evidences of loss of power in the extremities may be temporarily developed. Unless the case end fatally, consciousness is regained in four or five days. During convalescence the gait is staggering and often irregular, and deafness is complete. The staggering is usually recovered from, but the loss of hearing is permanent. Voltolini and Reichel believe that the symptoms are the result of *primary purulent labyrinthic otitis*, but other observers, notably Knapp, affirm that the disease is always secondary to meningitis or some septic fever. It is certain that in epidemic cerebro-spinal meningitis the inflammation occasionally extends to the labyrinth, and it is probable that the same thing sometimes occurs in other forms of meningitis. It seems to me, however, likely that there are cases in which inflammation of the labyrinth is primary.

the heart's action, which might readily lead to the supposition of cardiac diseases and cardiac vertigo.

Chronic kidney-disease does not very frequently give rise to vertigo, but I have seen pronounced vertiginous attacks the only decided symptoms of a mild uræmia. In one case, a woman, who eventually died in uræmic convulsions, the paroxysms of vertigo came on only when no food had been taken for three or more hours, and were for a long time supposed to be gastric. The attacks commenced with extreme pallor of the face, and the appearance of dark rings under the eyes; then the woman would speak very hesitatingly and slowly, and a moment later cease with a dazed expression of countenance. The mental confusion was so marked that she did not know where she was or what she was doing. After the attack she did not remember what had occurred during the paroxysm; but she never fell in an attack, and she would always give some response when spoken to. For a long time the attacks were at once relieved by giving a few mouthfuls of some hot drink.

Essential Vertigo.—There is a class of rather infrequent cases in which none of the known causes of vertigo can be discovered, and to which the name of essential vertigo, given by Dr. Ramskill, may be well applied. It is entirely possible that in some instances a hidden peripheral irritation or structural brain-change may be the cause of the symptoms; on the other hand, it is possible that in the brain there are centres connected with equilibration which are liable to suffer from functional or structural disease and thus give rise to vertigo. The recognition of a case of essential vertigo implies simply that every known cause has been looked for and not found.

CHAPTER V.

TROPHIC LESIONS.

Under the head of trophic lesions I shall consider those alterations of structure which are apparently dependent upon disease of the nerves or of the nerve-centres, or which are, at least, closely connected with, and subsequent to, such nervous affections. The discussion of the methods in which these trophic lesions are produced seems to me beyond the province of the present work, but it may be allowable to state my belief that the nervous system does exert a direct and immediate influence upon nutrition,—that is, upon the structure of the body. A functional act, whether of a gland or of a muscle, is nothing more or less than a nutritive act. It has been long proved that a nerve may directly so affect the nutrition of the muscle-fibre or of the glandular cell as to cause the one to contract and the other to secrete; *i.e.*, it has been long proved that the nutrition of the muscle and of the glandular cell may be directly influenced by the discharge of nerve-force, and that therefore there are trophic nerves.

For the purposes of clinical study trophic lesions are divisible into those which rapidly destroy all the tissues in their immediate course, and those which are not thus destructive. Lesions of the second class are for the greater part essentially slow and progressive, although included in the class are some acute lesions which are more or less strictly confined to a single tissue, which, however, they do not rapidly destroy.

ACUTE DESTRUCTIVE TROPHIC LESIONS.

The destructive trophic lesions are the Decubitus Acutus of Continental writers, or the rapid Spontaneous Eschar; the *mal perforans*, or the Perforating Ulcer; and Raynaud's Disease, or Acute Symmetrical Gangrene.

Decubitus.—The term Decubitus is an unfortunate one, which really refers to the position assumed by the patient in bed, but has

been transferred to the sore, formerly supposed to result solely from pressure due to the position of the bedridden patient. It usually attacks the sacro-gluteal regions, but it may appear in any portion of the body which is subject to a slight continuous pressure, and is not infrequently seen in the heels. The first warning consists of one or several erythematous patches, variable in extent and irregular in shape. The color may be rosy, but more frequently is dark red or even violet. It disappears momentarily upon pressure with the finger. In rare cases, and, according to Charcot, only when the spinal cord is involved, there is about the erythematous patch an apparently phlegmonous swelling, with sometimes acute pain. Within twenty-four or forty-eight hours vesicles, or bullæ, form in the central portions of the erythema. They are reddish or brown-colored, and contain a liquid sometimes colorless, but generally opaque and bloody. In rare cases, under careful management, the vesicles and blebs wither and disappear without further symptoms: usually, however, the elevated epidermis is torn or drops off, leaving a bright red surface with bluish or violet points or patches. There is now some swelling and sanguinolent infiltration of the tissue for some distance beneath the bared surface. In the course of a few hours the reddish surface becomes blackish, and a slough of variable extent forms. The whole buttock may thus melt down in the course of a few hours. Sometimes the process is arrested and the slough separates, but oftener the process continues, and, unless the patient die too quickly, the deeper muscles, with the nerve-trunks and arterial branches, are laid bare, and finally the bones themselves appear. Generally death occurs from exhaustion, but, according to Charcot, a secondary purulent affection with metastatic abscesses may follow upon the acute bed-sores, and in rare cases gangrenous emboli occur in the lungs or in other portions of the body.

Acute decubitus occurs in disease of the brain and of the spinal cord. In cerebral hemiplegia it is always upon the paralyzed side.

In 1876, A. Joffroy (*Arch. de Méd.*, January, 1876) attempted to show that in cerebral cases the eschar was always the result of lesions of the occipital lobe or of the optic thalamus. This, however, is not correct, as the sloughing bed-sore has developed after hemorrhage in the external capsule and corpus striatum (Broadbent, *Lancet*, 1876); after focal lesions in the convolutions

(De Beurmann, *Soc. Anat.*, March, 1876); after hemorrhage into the extra-ventricular nucleus of the striate body (Dusaussay, *ib.*, January 21, 1876); after softening of the sphenoidal lobe (Leloir, *Progrès Méd.*, 1879); and after various other lesions. Moreover, Charcot has reported four cases in which the occipital lobes or the optic thalami were the seat of the lesion without the production of the eschar. It would seem, therefore, that acute decubitus may follow lesions of almost any portion of the brain.

Brown-Séquard has demonstrated that if the spinal cord be divided half-way through in an animal, acute sloughing ulcers will develop in the sacral region, although the part is neither subjected to compression nor irritated by the urine. The most interesting fact in connection with this traumatic spinal decubitus is that the eschars are limited to the side opposite to the section. In man acute decubitus has been noted after hæmato-myelitis, acute myelitis, traumatic myelitis, fracture of the spine, etc. It appears to be especially connected with destruction of the central gray matter of the cord. According to the statistics collected by Prof. John Ashhurst, after fracture of the spine decubitus is prone to occur in direct proportion as the injury is low down. Sir Benjamin Brodie, on the other hand (*Med.-Chir. Trans.*, 1837, vol. xx. p. 148), affirmed that the sloughing bed-sore develops most rapidly when the lesion is high up.

Perforating Ulcer.—Under the name of perforating ulcer (*mal perforans*) is described a peculiar ulceration which usually, but not always, appears upon the foot, and especially affects the immediate vicinity of the metatarso-phalangeal articulations of the big and the little toe. Often there is but a single ulcer on one foot, but there may be as many as three ulcers, and in not rare cases both feet are symmetrically attacked. The perforating ulcer may appear upon the hand, and there is reason for believing that it may even affect the internal organs. Thus, M. L. Terrillon, in the *Bull. de la Soc. de Chir.*, 1885, p. 403, reports a case of posterior spinal sclerosis, with fulgurant pains in the hands and arms, in which there were symmetrical ulcers on the thumb and the index and median fingers of each hand. Some years ago, in a case of gouty dementia (*Trans. College of Physicians*, Phila., 1884–85), I saw a circular ulceration three-fourths of an inch in diameter, with smooth, sharp edges, in two or three days eat through the

septum between the vagina and the rectum. It is well known that not rarely after extensive burns rapidly-perforating ulcers pierce the coats of the stomach, or more usually of the duodenum, with fatal results.

The first symptom of the perforating ulcer of the foot is generally a severe pain. This prodromic pain may, however, be entirely wanting. A small hemorrhage or ecchymotic spot now appears under the epidermis; in the course of a few hours the skin detaches itself, or more frequently becomes excessively thickened into a large, dry, corn-like mass; a small slough soon separates, leaving the ulceration round, with sharp, acute edges, piercing, it may be, only through the skin, but usually to the deeper tissues, and in many cases reaching the articulation or the bone. Around the ulceration there is apt to be serous infiltration and swelling. In rare instances, especially if the patient be put to bed and carefully nursed, the perforating ulcer is recovered from without loss of bone: somewhat more frequently the patient escapes with the throwing off of small necrosed flakes of bone. In most cases, however, the bone becomes seriously diseased and a sinus forms. In this condition the lesion appears as a small aperture leading by a narrow sinus to diseased bone and surrounded by thickened superimposed layers of epidermis. The surface of the spot is usually cold and anæsthetic, the characteristic feature of the ulcer being its insensibility to irritants and its freedom from pain during rest. Walking may cause suffering; and the fulgurant pains of locomotor ataxia are very frequently present, but do not have their origin or focus in the ulcer. Erysipelatous inflammation or erythematous exudations are apt to occur. Under these circumstances the limb becomes greatly swollen and œdematous, and the attack may terminate in erysipelatous suppuration and death. Except in the rare cases in which the ulcer heals early, all the bones of the foot, and indeed all the tissues of the foot, become diseased. Not only is the joint that is in immediate relation with the ulcer apt to be affected, but all the small joints of the foot frequently take on an inflammatory action which ends in an anchylosis, or undergo ulceration and destruction, resulting in luxations and deformities. The nails of the foot usually become brownish, dry, greatly thickened, curved, and furrowed. In some cases there is a

marked increase in the growth of the hair and in the pigmentation of the leg, and the whole foot may be bathed in a peculiarly fetid sweat.

It has been denied that the connection of perforating ulcer with disease of the nervous system is other than accidental; but since the paper of MM. Duplay and Morat (*Arch. de Méd.*, 1873) it seems to have been almost universally acknowledged that the ulcer is the direct or indirect result of various nervous affections. The similarity between the perforating ulcer and the ulcerations of leprosy was, in 1871, strongly commented upon by Estlander, and in 1872 M. Poncet, in tracing the relations between leprosy and perforating ulcer, found that the nerves in perforating ulcer have their connective tissue increased and their fibrils atrophied. Duplay and Morat subjected the affected parts in six cases of perforating ulcer to microscopical examinations, and in each case found an advanced degeneration of the nerves. Morat (*Lyon Méd.*, March, 1876) reported a case in which the perforating ulcer followed traumatic section of the sciatic nerve. These observations have been confirmed by a number of observers (see Ross, 2d ed., vol. i. p. 259), and it would appear that perforating ulcer may be due to a disease of the nerve-trunks.

The great frequency of the affection in locomotor ataxia indicates, however, that it is not caused solely by lesions of the nerve-trunks. It is, of course, possible that the nerve-trunks are diseased in those cases of locomotor ataxia in which perforating ulcer occurs; but until this is proved we must consider that the perforating ulcer may be produced by various nervous diseases, of which the most important are posterior sclerosis and disease of the nerve-trunks. In locomotor ataxia this ulceration may be a very early symptom; and if in a case of mal perforans the knee-jerk be absent, the diagnosis of locomotor ataxia may be considered established, unless positive symptoms of disease of the nerve-trunks (such as tenderness) or of myelitis be present.

Raynaud's Disease.—Under the names of Dead Finger, Anæmic Sphacelus (Myrtle, *Lancet*, i., 1863), Local Syncope, Erythromyalgia (Mitchell), and Symmetrical Gangrene, there have been described by various writers groups of cases which are at present generally thought to represent a single disease, commonly known as Raynaud's disease, because the first clear recognition and

elaborate deseription of it were given by Dr. Maurice Raynaud (*L'Asphyxie locale*, Paris, 1862). The unity of these groups is not, however, entirely established.

In the most aeute form of the disease as deseribed by Raynaud the beginning of the attack is painless and sudden; the skin of the affeeted part becomes of a dead-white color, sometimes even a little yellowish, and appears entirely devoid of blood. Cutaneous sensibility is lessened or altogether destroyed, so that the fingers, which are the parts usually affected, may be pinched without pain: even when the sensation of eontact is entirely lost the power of distinguishing heat and cold may be retained. The temperature of the parts is very notably diminished; the power of movement is lost. After a time reaction sets in; the white eolor gives way to a eyanotic tint, which deepens to violet, and in some eases to a blaek eompared by Raynaud to that of a spot of ink. Pressure on the parts now produees whiteness, followed by instant return of eolor on removal of the pressure, showing that the diseoloration is owing to blood still inside of the eapillaries. The parts are at this time swollen. During the stage of reaction there is exeessive burning pain, which may begin even before the congestion, and in the height of the paroxysm rises to a prostrating agony. In cases of the severest type the loeal congestion soon deepens into gangrene.

Of the acute form of the affection described by him Raynaud makes three stages. The first is the period of invasion: it may last only for some hours, and is never protraeted beyond a month. The seeond period is charaeterized by intense congestion of the part; by the perpetual reeurrence of pain-erises, which usually pass off with an abundant emission of urine; and by the termination in gangrene, which is so rapidly developed that the local destruction is eomplete and limited in from eight to twelve days. The third stage is that of throwing off the gangrenous tissue, and is of variable length.

In the chronie type of the disease, aeording to Raynaud, there are frequent remissions, with violent attaeks, whieh may be provoked by exposure to eold, by a suppression of menstrnation, by fright, by a sudden emotion, or even by a mere momentary exeitement. This state may last for several years, and finally end either in gangrene or in reeovery without loss of strueture.

The term *local syncope* is applied to that condition in which the parts are excessively pale; the term *local asphyxia*, to the state of congestion.

Since the publication of Raynaud's article a number of cases have been reported upon the continent of Europe, in England, and in America. It is questionable whether the local asphyxia is not always preceded by the local syncope, and whether those cases in which there is no account of a local syncope ought not to be considered as a distinct group. It is certain that in many of the cases the local syncope had disappeared, if it had existed, at the time when the sufferers first came under medical observation: moreover, no history of its existence could be obtained. Some of Dr. S. Weir Mitchell's patients were so intelligent and so clear in denying a primary syncope that its existence is not probable. It was to cases of this character that Dr. Mitchell, believing them to be a distinct group, gave the name of erythromyalgia. On the other hand, in some recorded cases frequent attacks of syncope occurred in the earlier months of the disease, and finally gave way to a perpetual local asphyxia whilst the case was being watched by the physician. Thus, Dr. Calcott Fox (*Clin. Soc. Trans.*, vol. xviii. p. 305) details the case of a woman whose fingers for ten years suffered from frequent paroxysms of local syncope, but at last passed into a condition of continuous local asphyxia. In these chronic asphyxia cases the pain is increased by allowing the part to hang down, by warmth, by exertion, or by any act or position which naturally tends to increase the amount of blood in the affected member. During the condition of congestion or local asphyxia there is tenderness, which may be accompanied by excessive hyperæsthesia or may be revealed only by firm pressure. Gangrene may at any time come on, even in cases which have lasted for many years, but in the most chronic form of the affection other nutritive alterations are not rare: thus, in a case reported by Dr. Fox, occasional blood-blisters formed on the affected fingers, leaving raw surfaces which were slow to heal, and especially affected the edges of the nails. In such cases some of the fingers may have their phalanges completely atrophied and their nails shrivelled up, whilst in other fingers the ends become markedly conical, with their nails curved over them.

During the stage of syncope the local temperature is markedly abated: thus, M. Lannois (*Paralysie vaso-motrice*, Paris, 1880) has noted it 4.7° C. below that of the opposite side. During the period of congestion the temperature rises, and it has been noted as high as 19° F. above that of the opposite side (M. Allen Sturge). In one of Mitchell's cases, whenever the foot was suspended intense congestion came on, accompanied by excessive pain and by great rise in the temperature.

Erythromyalgia appears to affect children more frequently than older people. In many of the reported cases the patients had been previously of robust health; in some instances the neurotic temperament has been strongly expressed; and in a few cases the affection has developed during the progress of diabetes. In several more or less pronounced cases hæmoglobinuria has been present, sometimes accompanying the attacks of local syncope, in other instances occurring indifferently to them. In two or three cases ocular troubles have been noted, and once or twice disorders of audition. The reflexes have occasionally been increased, but generally have been normal.

Although the fingers are the parts most commonly affected, other extreme portions of the body are often attacked. The toes are frequently the seat of the disease, and in several of Mitchell's cases the gangrene affected a great portion of the sole of the foot. All the phenomena of the disease have frequently been observed in the ears, and in a few instances the end of the nose has suffered destruction.

The exact nature of Raynaud's disease is still obscure. The condition known as local syncope is probably due to an intense vaso-motor spasm, but the cause of such spasm has thus far eluded observation. That it is a general wide-reaching influence is shown by the implication of the ears, nose, fingers, and toes, and by the occasional hæmaturia. The occurrence of ischæmic aphasia (Weiss, *Zeitschr. für Heilk.*, 1882) strongly indicates that internal vascular areas, as well as those of the extremities, may suffer. No such condition as local asphyxia follows even the complete vaso-motor paralysis of nerve-section, and if it be really paralytic it can be explained only by supposing that the muscles of the walls of the vessels are so absolutely exhausted by over-effort that when relaxation follows their local tone is entirely lost.

Professor Pitrès (*Archives de Physiologie*, 1885, p. 106) found in one case of Raynaud's disease extensive peripheral neuritis, and Dr. A. Bidder* has reported (*Arch. für Klin. Chir.*, xxx. 810) a case in which gangrene of the fingers followed fracture of the arm with injuries to the nerve; but it does not seem probable, and certainly is in no way proved, that peripheral nerve-lesion is constantly present in the disorder.

Various facts indicate an obscure but close relationship between the dead fingers, scleroderma, and morphœa. In some cases of dead fingers a peculiar, raised, wheal-like eruption has been present. Further, the repeated coexistence of two such rare affections as scleroderma and Raynaud's disease (for cases, see Dr. C. Fox's article, *Clin. Soc. Trans.*, vol. xviii. p. 305; also Dr. Finlayson, *Medical Chronicle*, 1884–85, p. 315) cannot be accidental.

TROPHIC LESIONS NOT ACCOMPANIED BY WIDE-SPREAD DESTRUCTION OF TISSUE.

Trophic changes which are not accompanied by wide-spread destruction of tissue and are more or less confined to a single tissue are best classified for study according to the tissues affected. I shall therefore discuss—first, trophic changes in the skin and its appendages; secondly, trophic changes in the muscles; thirdly, trophic changes in the bones; and, fourthly, vaso-motor disturbances and disorders of secretion.

TROPHIC SKIN-CHANGES.

Skin-Diseases.—It is probable that many of the diseases of the skin are dependent upon, or at least connected with, affections of the nervous system. Thus, Jaining de St.-Just (article "Scarlatine," *Dict. Encycl.*, 3e série, vii. 307) relates the case of a hemiplegic in whom for two days the eruption of scarlatina was limited to the normal side, and Chevalier (*Thèse*, Paris, 1878) describes a case in which variola was confluent and hemorrhagic upon the paralyzed side but discrete upon the other; and in a case of M. Bouilly (cited by Arnozan), during an attack of smallpox

* Considering the possible lesion to blood-vessels, and the free use of the gypsum bandage, not much weight can be attached to this case.

no pustules appeared upon the leg the sciatic nerve of which had previously been divided. Our present knowledge of this subject is, however, so scanty that a most important field of research remains almost uncultivated. That desquamation may be effected by nervous influence is shown by a case reported by N. Bouilly (Arnozan, *Des Lésions trophiques*, Paris, 1880, p. 151): a man had a neuroma of the sciatic nerve, and below the tumor the epidermis was covered with small, dry, blackish or brownish scales, easily detached, and having an appearance like that of ichthyosis; after removal of the neuroma the skin resumed its normal appearance. Ballet and Dutil (*Progrès Méd.*, May, 1883) reported three cases of skin-alteration resembling ichthyosis in disease of the spinal cord, and M. Gautier one in a person suffering from lead palsy. Tischer has seen exfoliation of the skin follow the course of an inflamed nerve, and Schiefferdecker the skin thickened, scaly, brown, perpetually covered with malodorous sweat, and adorned by hypertrophic nails and hair. (See Ross, *loc. cit.*, p. 248.) The pigmentation of the skin which sometimes occurs in Raynaud's disease has already been pointed out, and it is probable that the bronzing which is characteristic of Addison's disease is due to nervous influence. According to Morselli, there is a peculiar form of vitiligo seen in the insane in which whitish spots surrounded by pigmented borders are more or less symmetrically arranged about the head and neck. Bourneville and Poirier (*Progrès Méd.*, 1879, No. 24) have reported somewhat similar discoloration of the skin in a person suffering from cerebral tumor, whilst Dumenil has noted pigmentary alterations following chronic neuritis. G. Rossolymmo (*Arch. für Psych.*, 1884, vol. xv. p. 723) has recorded a case of a person suffering from locomotor ataxia, the right half of whose forehead, cheek, and nose, etc., became covered with irregular sharply-bounded spots in which the hair turned white.

In 1831, Dr. R. Bright called attention to the possible etiological dependence of herpes zoster "upon distention of the sentient nerves." In 1853, Romberg (*Syd. Soc. Translations*, vol. i. p. 84) also noted the seeming connection between herpes zoster and intercostal neuralgia. In 1855, M. Delioux suggested that herpes zoster might be due to neuritis; and in 1859, M. Charcot published cases of herpes following upon a wound of the nerve.

These observations have been abundantly confirmed (see Ross, vol. i. p. 243), and it has been shown that neuritis may give rise to herpetic and other forms of eruption. The first stage of acute decubitus is a bulla, and pemphigus has been noted in various nervous diseases. (See Ross, *loc. cit.*, p. 247.) Papular and pustular eruptions after neuritis have been noted by Charcot and by Vulpian; whilst the quickness with which erythematous eruptions and urticaria are developed by certain forms of gastric irritation demonstrates that they are often nothing more than reflex nervous phenomena. I have seen a furious urticaria replace the chill-stage of a malarial paroxysm; Charcot reports a case of locomotor ataxia in which enormous wheals covered the parts through which the pains were darting; and ecchymotic spots not very infrequently appear during the pain-crises of posterior sclerosis.

In the elaborate studies made by Mitchell, Morehouse, and Keen of the results of gun-shot injuries of the nerves (see Mitchell, *Injuries of Nerves*), it was shown that in many cases the eruption following the nerve-injury is composed of small acutely-pointed vesicles, which may well be described as eczematous: eruptions of similar character have been also noted after injuries to the ulnar nerve (see Mitchell, p. 154).

The so-called "*glossy skin*" (*causalgia* of Mitchell) is a very curious alteration of the skin, which was first distinctly described by Mr. Paget, although noted as early as 1813 by Mr. A. Denmark. It occurs as the result of injuries to nerves and the consequent neuritis. The affected skin has the appearance of thinness, is very smooth, glossy, and shining, as though varnished, is usually deep red or mottled, or red and pale in patches, free from hair, and often looks as if it were tightly drawn over the tissues below. This condition of the skin is always associated with a horrible burning pain, which frequently rises to agony, and often precedes the nutritive changes. Over the altered surface come and go groups of vesicles, whose eruption is attended with a temporary amelioration of pain. Dr. Mitchell believes that this causalgia may result from central nervous disease; but the case upon which he appears to have based this opinion resembles so closely spinal meningitis with a descending neuritis that in the absence of an autopsy the diagnosis is exceedingly uncertain.

Hair and Nails.—Trophic changes frequently take place in the

hair and in the nails. The whitening of the hair which occurs during an attack of migraine will be discussed under the head of Migraine. The change which occurs in the color of the hair from nervous influence is very remarkable, and at present inexplicable. There is a prominent surgeon in this city the hair of whose head is said to undergo a distinct temporary alteration whenever he has a severe, trying surgical operation on hand. It is well established that the hair may, under emotional excitement, change its color entirely and permanently during the course of a few hours or a few days. One of the best authenticated of modern instances of such a phenomenon is reported by D. P. Barry, staff-surgeon in the British army (*Medical Times and Gazette*, April, 1859, vol. i. p. 367). Near the close of the Sepoy Rebellion a Bengalese was brought in and questioned previous to execution. While actually under observation, within the space of half an hour his hair became gray on every portion of his head, it having been glossy jet-black at the beginning of the examination. The attention of the by-standers was first attracted by the sergeant, whose prisoner he was, exclaiming, "He is turning gray!" Gradually but decidedly the change went on until a uniform grayish color was reached. The older records contain various reports of this abrupt *canities*. Thus, Ludovico Sforza, having been taken prisoner by Louis XII., his mortal enemy, was seized with such terror that the night before he suffered punishment his hair, which had been before very black, became very white, so that his guards the next day thought him to be another person. Montaigne comments upon a gentleman one-half of whose beard and one eyebrow suddenly became white in consequence of a violent emotion. Guarini da Verona suddenly turned gray when the loss at sea of the Greek manuscripts which he had with infinite toil collected at Constantinople was announced to him. Bichat (*Anatomie Générale*, iv. 815) and M. Rayer (*Traité des Maladies de la Peau*, iii. 733) each record a case in which the hair turned white during a single night; and a number of cases have been collected by J. Moleschott (*Physiolog. Skizzenbuch*, Giessen, 1861) showing that a similar alteration may take place more gradually in the course of a few days.

Peripheral nerve-lesions undoubtedly also affect the nutrition of the hair. In causalgia the hair usually falls out,—a result

which in animals habitually follows section of the nerve. On the other hand, both Pouteau and Larus saw the hair become coarse, hard, and stiffly erect in traumatic neuralgias. Bellingeri also noticed the hair becoming thicker and harder and growing faster, whilst in a case recorded by Hamilton, during neuritic symptoms following a lancet-wound, the arm became thickly covered with hair. (See Mitchell, *Injuries of Nerves*, Phila., 1872, p. 164.) In a case of arsenical poisoning, with wide-spread neuritis and complete degeneration of the muscular structure, which I watched for many months, the legs became covered with a thick growth of hair several inches long.

As was, I believe, first observed by Dr. Mitchell, the growth of the nails is habitually arrested upon the paralyzed sides in cases of *cerebral hemorrhage*. This is easily demonstrated by staining the nails of the two hands with nitric acid: frequently a lunate appearance of growth at the bottom of the nail is the first evidence of returning functional power. After total section of a nerve the nails are apt to become clubbed, and in rare cases painless whitlows are developed. In traumatic neuritis, especially in connection with causalgia, nails to which the affected nerves are distributed undergo remarkable changes. The alteration consists in a curve in the long axis and extreme lateral arching, and sometimes a thickening of the cutis beneath the end of the nail, whilst the skin is retracted from the base of the nail so as to leave a partially-exposed sensitive matrix. In certain cases of nerve-lesions the nails become dry, scaly, and cracked, and in others they undergo atrophy; sometimes, as in the case reported by Hayem, they fall out entirely.

As will be explained in discussing the trophic lesions of bones, it is not rare for the teeth to fall out in locomotor ataxia, on account of the destruction of the alveolar processes.

TROPHIIC BONE-CHANGES.

Peripheral Nervous Diseases.—Trophic alterations of the bones may be produced by disease of the nerve-trunks. M. Avezou (*Thèse*, 1879) has collected a number of cases showing that nerve-lesions can produce atrophy of the bones, and Lobstein details a case of a man who had an injury of the sciatic and crural nerves, in whom, after death, the femur on the injured side was

found to weigh only one-third that of the normal side. Ogle records a case (*St. George's Hospital Reports*, 1871) in which section of the median nerve was followed by wasting of the bones through the whole distribution of the nerve. It is well known that in poliomyelitis of the young arrest of development follows the alteration of the spinal cells. The *facial trophoneurosis* of Romberg is possibly of this nature,—an arrest of development of the bone following poliomyelitic atrophy of the facial muscles.* M. Bouchut (*Gaz. des Hôpitaux*, 1878, p. 629) has reported the case of a child, seven years old, in whom an ascending neuritis, the result of an injury, had been followed by marked lessening in the bones of the arm, as well as by arthropathies, of which it is uncertain whether they were trophic or rheumatismal.

Central Nervous Disease.—Many years ago it was noted that the bones of insane patients are frequently broken, and that multiple fractures are quite common. These constantly-recurring fractures have been brought forward by the opponents of insane asylums as evidence of cruel and rough handling on the part of attendants, but it is now proved that the causes of the accidents lie chiefly in the bones themselves. Under certain circumstances the bones of persons suffering from general paralysis, and probably the bones of those suffering from other forms of insanity, become enlarged and brittle. When broken, these bones unite easily and very rapidly, with the formation of an excess of callus, so that there may be a large tumor at the seat of fracture. After death in such cases, it will be found that the bones are notably enlarged; that they are so soft as to be readily cut with a knife; and that there exudes from the cut surface a pink or reddish sanguinolent juice, which, when placed under the microscope, is found to contain large quantities of nuclei and imperfectly-developed cells. On section, such bone will be found to be more porous than normal, and the microscope will reveal great dilatation of the Haversian canals, which are filled with a fluid containing embryonic cells. A very curious feature in this form of bone-disease is that it attacks almost exclusively the skeleton of the trunk. It is the ribs, the bones of the pelvis, or the vertebræ that are affected.

* For an account of this affection, see Frémy, *Étude critique sur la Trophonévrose faciale*, *Thèse*, 1873; also Lande, *Aplasie lamineuse progressive*, *Thèse*, 1870.

Dr. Moore is stated to have made a chemical analysis of these bones, and to have found a remarkable lessening of their inorganic matter. I have seen one case in which an osteoporosis like that of general paralysis existed in a patient who was supposed to be suffering only from chronic alcoholism. As, however, I saw the man but once during life, and as the history was very imperfect, the diagnosis may have been erroneous.

TROPHIC CHANGES IN JOINTS.

The most important of the changes which are produced in bony tissues by diseases of the nervous system are those which are connected with alterations of the joints.

Hemiplegic Arthropathies.—As was especially pointed out by Prof. Charcot, there is a peculiar form of arthritis connected with hemiplegia which is especially apt to occur when the paralysis is dependent upon minute foci of softening. Very often this form of arthritis is supposed to be due to rheumatism, and the patient is believed to be suffering from another disease—acute or subacute rheumatism—supervening on the attack of hemiplegia. The diagnosis between trophic and rheumatic arthritis is to be made by attention to the following particulars: first, the hemiplegic arthritis develops about the time at which late muscular contractures usually come on; secondly, the trophic inflammation of the joints, at least in the early stage, is limited to the affected side; thirdly, the pain may be moderate, but the tenderness is excessive; fourthly, the swelling, which is pronounced, develops rapidly, and is accompanied by distinct œdema, with pitting on pressure.

The history of hemiplegic arthritis differs entirely from that of chronic rheumatism. In rheumatic arthritis there is no tendency to the development of pus, and little or no tendency to the breaking down of bony tissue, the effusion within the joint being serous and remaining so for months. The cartilages may be removed, but the bone beneath the cartilage becomes hard, thickened, and of irregular growth, and has very little tendency to ulceration and destruction. Frequently osteophytes are found in the effusion. In the joint affected with hemiplegic arthritis, although the process is very slow and the arthritic changes may continue for months and even years, there is a tendency to the formation

of purulent liquids, and to the destruction not only of the cartilages but also of the bone.

In a patient of my own there was complete hemiplegia on the right side, with aphasia. She complained greatly of pain in the leg and arm. The moment she thought the limb was to be examined she would scream with the mere fear of contact: when the joint was touched, the emotional disturbance became uncontrollable. The joints were swollen, very glossy, and hard to the touch. When she first came under observation the affection was confined to the hemiplegic side, and was evidently not rheumatic. There was no history of rheumatism, and the joints did not present the peculiarities of rheumatic joints. For a year or more the joints remained in the same condition, but finally those of the other side became slightly affected. After death the original lesion of the brain was found to have been a large hemorrhage in the neighborhood of the claustrum, entirely destroying the external capsule: hence the complete hemiplegia and aphasia, and the trophic lesions. On opening one of the joints, I found that it contained a moderate amount of purulent serum, that the cartilages were almost entirely destroyed, and that the surface of the articulations was largely affected and eroded. After the bones were boiled, the articulating surfaces were found to be exceedingly porous, and in some places part of the bone had been eaten away. The bones of the arm were very light, owing to the thinness of their shafts. There had been not only a destruction of the joint, but also an atrophy of the shaft of the bone.

Spinal Arthropathies.—Changes in the joints which must be looked upon as trophic are not rare phenomena in locomotor ataxia. They belong among the prodromic symptoms, usually being developed after the fulgurant pains, but before marked disorder of co-ordination. In rare instances they are of diagnostic importance. In any case their presence, associated with loss of knee-jerk or with fulgurant pains, would be sufficient for the diagnosis of incipient locomotor ataxia. Sometimes, though rarely, they are developed in advanced stages of the disorder, but almost invariably under these circumstances they affect an upper extremity, and therefore really represent the early changes in the spinal cord, up which the disease is ascending.

Dr. M. Ball (*Des Arthropathies consécutives*, Paris, 1869) shows

that in its typical development there are three stages of sclerotic arthropathy. In the first stage the joint suffers from hydrarthrosis. The effusion is serous, and never contains blood, pus, or albuminous flocculi. It is not limited to the articular cavity, but distends the bursa or the fibrous tissues around the joint, and may, indeed, involve for a considerable distance the whole leg. The joint at this time is enormously swollen, hard, usually pale, and so resistant as not to pit on pressure. The amount of fluid which it contains is very large. Thus, in a case reported by Dr. Ball, three hundred grammes of liquid were taken out of the joint by three successive punctures. There is no inflammation of the joint. At a post-mortem examination made by Dr. Ball, the synovial membrane did not show any abnormal vascularity, and there were no vegetations in the articular cavity. In rare cases the effusion is absorbed, but usually the second stage is soon developed. At this time the joint is much swollen, hard, and bony, with an evident increase in the size of the bony surfaces. In the third stage there is destruction of the articulating surfaces, and in some cases so much absorption of the bone and changes in the ligamentous structure as to produce great alterations in the power of movement. The epiphyses especially undergo atrophy and change; the ligaments are elongated, probably as a consequence of prolonged stretching by the excess of fluid, and at last a condition of subluxation or perhaps of complete luxation of the joint occurs, so that the ataxic may be able voluntarily to put out of joint a shoulder, a knee, or other joint without pain, though marked grating can be felt during movement. In a case reported by M. Oulmont, the patient was able to bend his leg in such a manner that the sole of the foot could be placed upon the internal surface of the thigh.

Tabetic joints usually develop with great rapidity, and in most cases without apparent cause. The patient will go to bed in the evening with the joint seemingly in its normal condition and wake up with it swollen in the morning. There are recorded cases, however, in which these arthropathies followed exposure to damp or slight traumatisms. In several of my own cases the patients insisted that they had broken the foot or ankle during some moderate exertion. It is probable that in these instances a slight sprain was followed in the course of a few hours by immense exudation.

Usually in the beginning of, as well as later in, the attack, the joint is not red, and there is little or no discoloration; but M. Michel speaks of having seen great enlargement of the veins, and even rupture of the large saphenous vein, which caused the whole leg to turn black. In one of my cases a similar blackening of the limb was asserted to have accompanied the first development. It is doubtful whether there is ever any fever or local heat, although M. Ball reports one or two cases in which fever was said to have existed in the beginning; but, as no thermometric studies seem to have been made, there is considerable doubt as to whether the general temperature was really elevated.

MM. Charcot and Bouchard have noted in two instances a peculiar articular creaking or crepitus, preceding by some days the serous exudation.

Spinal arthropathies are most commonly observed during the second stage of M. Ball: such joints are large, very hard, evidently containing much water, and at the same time having an increase of their composite bones, are perfectly indolent, and free from redness or heat, although attempted movements usually cause pain. In one of my own cases, in which it was doubtful whether the alteration of the joint should be considered hysterical or ataxic, there was marked hyperæsthesia. It is possible that such hyperæsthesia may be looked upon as a diagnostic means of distinguishing between the hysterical and the ataxic joint. From the rheumatic joint the ataxic is at once separated by the absence of heat, excessive tenderness, and pain. Care is sometimes necessary not to mistake the fulgurant pains of the locomotor ataxia, which may dart and play about a joint, for the true joint-pains of a rheumatism.

The ataxic arthropathy is sometimes unilateral, but is very frequently more or less symmetrical. It attacks especially the knees, and next in order of frequency the other joints of the lower extremities, but it may occur in any joint of the body. In eighteen cases collected by M. Ball, the knees were affected in eleven cases, the shoulder in three, the coxo-femoral articulation in one, the metacarpo-phalangeal joints twice, and the elbow once. The small joints of the foot are frequently attacked, giving rise to a peculiar deformation to which the name of the tabetic foot (*pied tabétique*) has been given by Prof. Charcot. The outer border of

the foot is often enormously thickened, so that the inner border does not touch the ground. A peculiar impression of the foot is thus produced, like that of the accompanying drawing, reproduced from a paper in *Bull. Soc. des Hôp.*, Paris, November 4, 1885.

Fig. 6.



In a case of this character of my own the arch of the foot was entirely lost, so as to bring the plantar surface continuously to the ground from the heel to the toes. The change had been accompanied by an increase in thickness over the tarso-metatarsal articulation, so that, although the foot rested flat upon the ground, this region was very prominent. The prominence was most marked on the inner edge. The deformity was greatest in the left foot.

Fig. 7.



At the autopsy in my case, besides advanced posterior spinal sclerosis, the following condition was found:

The ankle-joint, which was the first examined, exhibited no enlargement, nor did the articulating surfaces of the tibia and

fibula yield any evidences of disease. The articulating surfaces of the astragalus, however, were here and there denuded of cartilage and much roughened. Similar changes, though slight, were noticed in the calcaneum, but it was in and about the joint formed by the internal cuneiform and first metatarsal bones that the greatest amount of change had occurred. The cartilage had entirely disappeared from its upper portion. Here the two bones had become firmly united. The microscope revealed a continuous osseous structure from one to the other. The lower portion of the joint, which was equivalent to about three-fourths of its entire area, was filled by continuous or adherent surfaces of cartilage, while here and there a narrow chink, representing the original cavity of the joint, was left. The bones appeared enlarged and distorted, and an examination of their internal structure showed that the cancellated tissue had been replaced here and there by small masses of dense ossific deposit.

The middle cuneiform and second metatarsal bones were partly crowded over the internal cuneiform and first metatarsal bones, and presented lesions similar to those just described. The heads of the two metatarsal bones had in one place become continuous, and one section revealed an isthmus of bone uniting an angle of the internal cuneiform with the head of the second metatarsal.

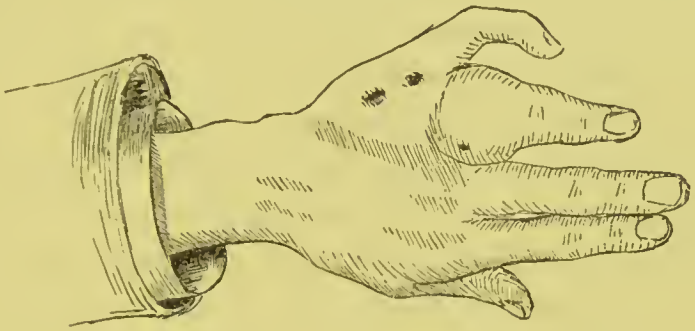
The general impression given by the study of these lesions was that of a mass of bones which, being at one time softened, had been mechanically distorted and displaced. The absorption or deposition of bony tissue appeared to follow no rule, nor did the destruction of the cartilage distinguish itself by any peculiarity other than that it seemed to precede the changes in the bones.

Much more rare than the tabetic foot is the tabetic hand, of which I have never seen an example. The cut on the opposite page, after Ball, indicates that it is scarcely less characteristic and peculiar than is the *pied tabétique.*

The portions of the bone most prone to be attacked in locomotor ataxia are the epiphyses: although first much enlarged, they finally undergo atrophy, which may reach such an extreme that only the traces of the head of the bone can be found, surrounded very frequently by long stalactitic points. Although Arnozan seems to deny it, and there are very few, if any, autopsies to prove it, the clinical evidences show very clearly that the stage

of atrophy is preceded in most if not in all cases by one of hypertrophy. M. Liouville (quoted by Arnozan) found in one case of scapular arthropathy dilatation of the Haversian canals, which were filled with embryonic cells and fatty matter. M. Regnard, in making an analysis, found the osscine in normal quantity, but the

Fig. 8.



phosphates very remarkably diminished and the fatty matter enormously increased. These histological and chemical results show a strongly-marked resemblance between the bones of locomotor ataxia, of osteomalacia, and of general paralysis.

Not rarely in locomotor ataxia the shaft of the bone is also attacked, and fractures from muscular exertion during life are in such cases very common. Although in various post-mortems the shafts of such bones have been found much atrophied and very hard, yet there is reason for believing that the pathological process is not essentially different from that which occurs in general paralysis. In cases of fracture there is an enormous and excessively rapid formation of callus, whilst in some instances portions of the bone have been found hard and atrophied and other parts increased in size and spongy. Again, one femur has been found atrophied and its fellow enlarged and in a condition similar to that which occurs in general paralysis. It would, indeed, appear as though various diseases of the nervous system produce changes in bone which may end in atrophy and hardness or may result in a permanent production of a condition allied to osteomalacia; since not only have the alterations which have just been described been met with in general paralysis and in locomotor ataxia, but Lagrange (*Thèse*, 1874) has reported a case of scleroderma with neuritis in which the phalanges were extremely atrophied and their articular surfaces lost, while the microscope

revealed structural lesions very similar to those which occur in general paralysis and locomotor ataxia,—namely, engorgement of dilated Haversian canals with fat-granules and embryonic cells. There is also reason for suspecting that similar changes in the bones occur in leprosy, although close studies are at present a desideratum.

Loss of Teeth.—A curious result of trophic bone-changes which is not very rare in locomotor ataxia is a rapid loss of the teeth. This occurs entirely independently of the condition of the teeth and gums, which may be perfectly sound and free from all soreness. The attention of the patient is suddenly awakened by the teeth becoming loose and dropping out one by one at intervals, sometimes so rapidly that all the teeth of one jaw are lost in the course of a few hours or days. It is rare for the two jaws to be simultaneously attacked. In some instances a shedding of the teeth *en masse* has occurred during sleep and threatened strangulation. The edentulous jaw-bone continues to waste until it is reduced to a mere shell. Although there is no soreness in the gums or teeth, it almost invariably happens that for many months or even years preceding the lesion the patient suffers from violent pains about the face. These pains are shooting, and, even though they occur in regular crises and are associated with loss of sensibility, they are usually supposed to represent simple trigeminal neuralgia. They are, however, the fulgurant pains of locomotor ataxia, and are caused by the involvement of the nucleus or roots of the trigeminal nerve in an ascending posterior sclerosis. The falling of the teeth is due to the destruction of the alveolar processes by progressive trophic changes, which in the jaw-bone proceed in a manner entirely parallel with those of the long bones.

If posterior sclerosis begins in the upper portion of the spinal cord, the loss of the teeth occurs early in the affection, because it represents the stage preceding loss of co-ordination. When, however, as is usually the case, the degeneration of the spinal cord is an ascending one, it may not reach the trigeminal nucleus until late in the general disorder.

Artificial Spinal Arthropathies.—The only attempts to produce by operations upon the nervous centres bone-lesions similar to those of locomotor ataxia which have been crowned with any

success have been those of Dr. Giacomo (*Soc. de Biologie*, March, 1885, p. 156). This investigator cut in a very large dog the posterior roots of three lumbar nerves between the ganglia and the cord. After some months the joints of the left foot became enormously swollen and œdematous, without increase of sensibility or of temperature. At the autopsy it was found that degeneration had occurred in the spinal cord, and that the lesion of the joint corresponded in position with the secondary spinal alterations.

TROPHIC LESIONS OF MUSCLES.

Of all the trophic disturbances, the most important to the practical neurologist are those which occur in the muscles. The ganglionic cells immediately connected with the nutrition of the muscles are grouped together in the anterior cornua of the spinal cord. In any nervous disease, so long as the spinal cells and their connection with the paralyzed muscle are intact, no rapid change occurs in the structure of the muscle. Whenever there is a destruction of the spinal cells or an interruption of their pathway along the nerve, the muscle at once begins to undergo degeneration, and in from five to ten days such change is readily demonstrated. The early appearance of trophic lesions in a paralyzed muscle, therefore, proves that the lesion is situated either in the ganglionic spinal cells or in the motor nerve-trunk.

The detailed discussion of the anatomical changes in the muscles is foreign to the intent of the present volume. Suffice it to state that atrophy with granulation of the muscle-fibre is first apparent, then distinct fatty degeneration of the muscular fibres, with marked proliferation of nuclei, and finally a replacement of the muscle-fibre by cellular tissue, until at last the muscle is reduced to a fibrous band.

When a muscle is degenerating for want of spinal influence, it first loses its power of responding to rapidly-interrupted faradic or chemical currents, then to slowly-interrupted faradic currents, then to slowly-interrupted chemical currents, and lastly to slowly-reversed chemical currents. At this time occurs with the galvanic current the so-called *reaction of degeneration*, first discovered by Brenner, and since elaborated by Ziemssen and by Erb. To comprehend this reaction it must be remembered that it is obtainable

only by applying the electrode to the muscle. This is evidently connected with the fact that a muscle artificially separated from its nerve does not readily respond to the faradic current, although its answer to the slow galvanic current is the same as is that of the nerve.* According to some authorities, the separated muscle, before it gives the reaction of degeneration, responds more slowly than normal to the current: this is the so-called *modal* change. Even at this time the muscle may respond to milder currents than it normally notices. The divided nerve rapidly loses its power of answering the faradic current; to the galvanic current it is often at first abnormally sensitive, but soon its power of response declines to extinction. These quantitative changes are not attended with any qualitative alterations: the formula remains unchanged throughout.

In accordance with what has just been said, if the electrode be applied to a nerve-trunk of a degenerating muscle, it will be found that reaction is diminished in quantity but not altered in quality. When a galvanic current of very moderate strength is used, and

* I have never experimented with the action of galvanic and faradic currents upon isolated muscles, and authorities are somewhat at variance in regard to such action. Hughes Bennett affirms (*Electro-Diagnosis*, p. 36) that muscles have no true faradic excitability. Erb, on the other hand (*Electro-Therapeutics*, p. 76), states that the muscle has its own irritability. The nerve-endings in the muscles are so closely associated with the muscle-fibre that it is not possible by any mechanical procedure to separate one from the other, and any galvanic or faradic current which is thrown into the muscle must act upon the peripheral nerve-filaments. By the action of curare we are, however, enabled to paralyze the motor nerve-ending, and physiologically to isolate the muscle-fibre without injury to it. Under these circumstances, according to Landois and Sterling, faradic contractility of the muscle-tissue is much diminished, but not lost. It would appear that the muscle-fibre is capable of responding to any form of electricity, but is much more sluggish than is the nerve-tissue. Hence it responds more slowly, and it is necessary for the current to continue for a certain length of time in order for any response to occur.

There is no essential difference between the faradic and the galvanic current. All the chemical effects of the galvanic or chemical current are produced by the induced current obtained from the magneto-electric dynamos. The muscle being less sensitive than the nerve simply requires more time for the reception of the impression. On account of the exceeding brevity of faradic currents, it responds less readily to them than does the nerve. It responds just as badly to the galvanic or chemical current, provided such current be very rapidly interrupted.

the negative pole (cathode) placed over the normal muscle, but not over its motor point, a strong contraction occurs at the closure of the circuit; when, however, the positive pole (anode) is placed over the normal muscle, the contraction is much less; in neither case is there any contraction when the circuit is broken: in other words, with the normal muscle and a feeble current we obtain good cathodal closing contraction, slight anodal closing contraction, and no motion whatever at either cathodal or anodal opening. When a current of sufficient power is used, opening contractions are produced, and the anodal contraction is greater than the cathodal. The "reaction of degeneration" consists merely in a more or less perfect reversal of the above formula. The anodal (positive pole) closure then causes a stronger contraction than the cathodal (negative pole) closure. When there is only a slight degree of degeneration present, there is a correspondingly slight increase of anodal closing over cathodal closing contraction. A minimum degeneration would be indicated by an equality of the two closing contractions.

These alterations in the electrical reactions of a degenerating muscle are readily formulated, and in this way perhaps will be more readily grasped by the student. The symbols are as follows: An Cl C represents anodal closing contraction; An O C represents anodal opening contraction; Ca Cl C represents cathodal closing contraction; Ca O C represents cathodal opening contraction: $<$ represents is less than; $>$ represents is more than (the point of the $<$ being towards the lesser quantity).

Then the formulas are:

An Cl C $<$ Ca Cl C
An O C $>$ Ca O C } muscle normal.

An Cl C $=$ Ca Cl C
An O C $=$ Ca O C } muscle in first stage of degeneration.

An Cl C $>$ Ca Cl C
An O C $<$ Ca O C } muscle in more advanced stage of degeneration.

After the reaction of degeneration (D R of some authors) has been established, if the muscle continue to undergo change, the galvanic irritability slowly diminishes, stronger and stronger currents being required to produce an effect. When a certain stage is reached, all reactions cease, save a feeble An Cl C, and at last this is lost, and the muscle does not respond at all. When recovery

occurs, the electrical reactions of the muscle pass upward along the pathway they have descended.

The practical importance of the reaction of degeneration is greatly lessened by the circumstance that its demonstration usually requires much skill and patience, and that it probably is never present when a muscle retains its integrity as regards the faradic current. For the purposes of diagnosis the failure of response to the rapidly-interrupted faradic current is usually a sufficient test of the condition of a muscle. When a muscle loses its power of responding to the rapidly-interrupted faradic current in a week or ten days after the occurrence of paralysis, whether the reaction of degeneration can or cannot be satisfactorily demonstrated, the inference is positive that trophic changes are taking place in the muscle. If a few days later such muscle is unable to respond to any faradic current, this inference becomes a certainty. For the purpose of prognosis the study of the reaction of degeneration may be necessary, but it will, according to my experience, often be found disappointing.

Diseases which cause Muscular Degeneration.—A sudden loss of power in the muscle followed by rapid trophic changes must depend upon an interruption of the pathway between the spinal cells and the muscle, or upon disease of the cells themselves. The pathway may be interrupted by traumatism, or by neuritis of an acute and violent type, whilst the cells may be acutely diseased as the result of a violent and general inflammation of the cord, or as the result of the affection known as poliomyelitis (idiopathic or toxic), in which the ganglionic cells alone are involved. The diagnosis of the traumatism must be made out by the history: an acute myelitis is readily recognizable by the numerous symptoms which attend it (see page 65), and which dwarf the mere wasting of the muscle and loss of power. A general neuritis producing palsy and trophic changes is almost invariably attended with violent pain and tenderness; and even in the rare cases in which no severe pain is felt, the nerve-trunks are from the first excessively tender. In poliomyelitis the nerve-trunks are never tender. For the further consideration of the symptoms of multiple neuritis, see page 56.

The difficulties which offer themselves in the diagnosis of *idiopathic poliomyelitis* are usually confined to the early stages

of the acute form, when it is liable to be mistaken for an acute constitutional disorder. Poliomyelitis of an acute or subacute type occurs especially during childhood, although it may happen at any age. Its tendency to attack early in life is probably due to the fact that at this time the trophic cells are in an habitual condition of intense functional activity and excitement, because they have not only to maintain nutrition, but also to direct development.*

For the purposes of description it is necessary to recognize two types of poliomyelitis,—namely, the acute and the subacute,—although in nature these two types pass into each other by insensible gradations. In the most severe acute cases constitutional symptoms are often very violent and are apt to mask the characteristic local symptoms.

Laborde considers acute poliomyelitis as consisting of four stages: in this he is followed by Grasset; and if it be understood that these stages grade into one another and are arbitrarily created or separated for the purposes of description, the division is a good one. The first stage is that of attack; the second, that of more or less complete and generalized paralysis; the third, that of remission and localization of the paralytic phenomena; the fourth, the period of muscular atrophy and deformity.

During the first stage and in very severe cases the constitutional symptoms are most prominent. They consist of fever, disturbed cerebration, and convulsions. The fever is usually of short duration, lasting, it may be, only a few hours, sometimes a single day, but more rarely as long as one or even two weeks. It may be of a continuous type, but is very prone to be remittent, especially when it endures for any length of time. Even before the attack great nervous irritability, emotional excitement, and paroxysms of terror and other evidences of disturbed cerebration

* Acute poliomyelitis as it occurs in adults has been considered by many writers as a distinct affection; and even so clear and systematic an author as Grasset still devotes a separate chapter to its consideration. The acute spinal paralysis of adults is, however, without doubt the same disease as the essential palsy of childhood, or infantile paralysis. The old names should be abandoned, and whether it occurs late or early in life the disease should be known as poliomyelitis.

are often present; and when the fever develops, confusion of ideas passes into delirium, and slight somnolency into profound coma. At the same time convulsive manifestations appear. These may be local, but are more usually wide-spread: when generalized, they may consist of twitchings or of frequently-repeated spasmodic contractions, or may rise to the severity of the most furious general convulsions, which are often accompanied by vomiting, but are very rarely, if ever, fatal. It is possible, however, that some fatal obscure convulsive attacks in children really represent incipient poliomyelitis, the recognition of which may be impossible owing to the early death.

So soon as the paralytic phenomena become prominent the patient may be considered to have entered upon the second stage: at this time the fever usually, but not always, abates. The paralysis may be general, attacking the trunk and neck and all the extremities, and if at the same time the loss of power be complete, the child is unable to move any portion of the body below the face. Generally, however, it will be found that a leg or an arm or some portion of the body is only partially affected. Even in cases of most complete paralysis the sphincters are not implicated, although urinary incontinence is not rare; and in the severest cases there is no tendency to the formation of acute bed-sores. It is the exceptional case in which the whole body is paralyzed. As has just been described, paraplegia is more frequent than general paralysis; hemiplegia and simple monoplegia are at this stage alike very rare.

Trophic changes in the muscles can usually be detected by the sixth or seventh day, sometimes as early as the fifth, rarely not until the eighth or ninth.

The first decisive evidence of such changes is loss of faradic contractility and the appearance of the reaction of degeneration. In those muscles which are to recover their power, trophic changes occur very slowly and only to a slight extent. It is a sufficiently accurate rule for the purposes of ordinary prognosis to say that those muscles in which the reaction of degeneration is pronounced at the end of the eighth day will in all probability never recover their integrity, but that those which, although more or less completely paralyzed, respond to the faradic current at such time will probably regain their functional power.

The third stage of the disorder is that of remission and improvement. The paralysis begins in certain parts of the body to improve slowly: if the improvement involves equally, or nearly equally, all the muscles, the prognosis becomes very favorable, as the case usually goes on to complete recovery. In the majority of instances, however, certain groups of muscles do not regain power along with the general system. Indeed, the reaction of degeneration in these muscles may become more and more pronounced, and steadily advance, whilst electro-contractility in the other muscles is becoming normal. The paralysis usually ameliorates first in the neck and trunk, then in the arms, finally in the lower extremities. In rare cases the legs improve before the arms. Under such circumstances almost invariably many of the muscles of the arms settle into a permanent paralysis. In the majority of instances the final loss of power is confined to the lower extremities, and in them only certain groups of muscles remain paralyzed. The groups which are least apt to escape are the anterior and external muscles of the leg, and even more frequently the abductor muscles of the foot, especially the peroneal muscles. The muscles of the foot itself are rarely permanently paralyzed; the gastrocnemius occasionally. Of the muscles of the upper extremity the deltoid is the most likely to suffer. It is very exceptional for the muscles of the neck or of the trunk not to recover, although the erector spinæ muscles do sometimes atrophy.

The fourth stage of acute poliomyelitis which is recognized by Laborde and Grasset is really not a part of the disease at all. It is the state of paralysis that follows the disease; it is the permanent condition into which the patient is thrown by the disease. The muscles are atrophied: in rare instances their wasting is masked by fatty deposits. If the patient has been attacked early in life, the bones are arrested in development, so that the limb remains not only much smaller but also much shorter than its fellows. That the ligaments also suffer trophic changes is indicated by the complete relaxation of the joints.

The picture which has just been given applies to the acutest form of poliomyelitis. In the greater proportion of cases which are met with, the onset is far more insidious. The febrile symptoms may be overlooked, on account of their brevity and their

mildness; but it is probable that in many cases they are entirely wanting. Certainly in the majority of the many cases that I have seen, no history of the fever could be obtained. Under these circumstances the paralysis is usually not general or widespread; moreover, its development seems to be comparatively slow, so that not rarely at first only the gait of the child is affected, but after some days the paralysis deepens into a complete loss of power. It is probable that in these cases the original attack is limited to a certain number of spinal cells, and is not sufficient to produce constitutional disturbance, such as fever, delirium, etc., and also that in the affected cells some days are required for the full development of the degenerative process.

Arsenical Poisoning.—A set of symptoms closely resembling those of acute or subacute poliomyelitis are sometimes produced by metallic poisoning. It is probable that various metals are capable of causing these effects, but I have never seen them except as the result of arsenical or of lead poisoning. In arsenical poisoning they are usually preceded by such acute and characteristic manifestations as to make the recognition of their nature very easy; but in one or two cases of saturnine disease that have come under my notice the poliomyelitic symptoms were not preceded by any of the ordinary characteristic evidences of lead-poisoning. There must be a lesion either in the trophic centres or in the nerve-trunks, or more probably in each of these tissues. For an elaborate discussion of this question the reader is referred to my treatise on Therapeutics.

In the cases of arsenical poisoning with poliomyelitic symptoms which have come under my care the muscular atrophy has been associated, at least in its earlier stages, with violent darting pains, much tenderness, and loss of sensibility,—symptoms all pointing to the presence of neuritis; the falling away of the muscles is rapid, requiring, however, some weeks for its full development, and is associated with the presence of true reaction of degeneration. Although all portions of the body may suffer, the distal ends of the extremities are most prone to be attacked, and the legs preferably to the arms. In an elaborate monograph (Paris, 1881) Imbert-Gourbeyre has shown that atrophic arsenical palsy may take on a paraplegic or even a hemiplegic form, and may also simulate a multiple paralysis, to which last variety of palsy

it may indeed be considered to belong. According to Imbert-Gourbeyre, ninety-seven per cent. of the cases recover.

Lead-Poisoning.—In my experience, when symptoms resembling those of acute poliomyelitis have resulted from lead-poisoning, the upper extremities have been usually primarily attacked, the flexors and extensors both being attainted, and the alterations of the deltoid muscles following very rapidly upon those in the forearms. In several cases from the arms the paralysis has spread throughout the whole body, until, in the course of a few days, almost all the voluntary muscles have been involved, and in extreme cases the patient has at last become unable to do more than turn the head on the pillow. The affected muscles waste rapidly, offering the reaction of degeneration; pain and disturbance of sensibility have been absent, so that the picture has very closely resembled that of ordinary infantile paralysis. The successive implication of all the muscles occurring in an adult is, however, sufficient to raise a suspicion of toxic origin, and in all my cases the blue line upon the gums revealed at once the nature of the affection. Undoubtedly, however, this saturnine atrophic palsy may exist without the blue line. Under these circumstances the diagnosis can be positively determined only by finding lead in the urine. A symptom which in severe cases separates the toxic from the idiopathic disorder is the involvement of the sphincters, which are never attacked in idiopathic poliomyelitis, but are very apt to be paralyzed in acute saturnine atrophy of the muscles. The diagnosis is a matter of great importance, because all my saturnine cases have yielded to treatment.*

Progressive Muscular Atrophy.—Chronic wasting, with loss of power, of muscles occupying a more or less extensive territory of the body, is usually due to the disease known as progressive muscular atrophy, an affection which might be considered to represent the slow form of acute poliomyelitis, and therefore be named chronic poliomyelitis. We have, however, no positive knowledge as to whether the lesion is essentially the same in the acute and

* Although therapeutics are not at present under consideration, it is perhaps allowable to call attention to the extraordinary power over saturnine palsy possessed by enormous doses of strychnine. Of course the iodide of potassium should be administered to aid in the elimination of the lead, but never in the same prescription with the strychnine.

the chronic disorder. We know only that the lesion in each affection occupies the same site,—namely, the spinal ganglionic cells. The difference is that in acute poliomyelitis or infantile paralysis large masses of cells are attacked simultaneously, while in the chronic progressive muscular atrophy individual cells are affected, one after the other.

The onset of progressive muscular atrophy is always very slow and insidious. In most cases, before any marked change can be noted in the muscle, the sufferer perceives a loss of endurance, so that the part tires easily, or there may even be absolute loss of power for short exertion. Careful examination will now show, even if there be no sensible wasting, that the muscle is softer and more flaccid than normal. Sensibility is not impaired.

A symptom which often precedes any marked change in the volume of the muscle is fibrillary contractions. The variation in the amount of the fibrillary contraction is indeed so excessive as to lead to the suspicion that possibly the diseased process which attacks the ganglionic cells is not always the same, so that two or more diseases are comprised in progressive muscular atrophy as at present recognized.

In their mildest form the fibrillary contractions consist of slight irregular twitchings, occupying now this, now that portion of the belly of the muscle, and producing no effect except a corresponding movement of the skin over the contractions. The fibrillary contractions in their severest manifestations may amount to stormy peristaltic movements, hurrying through the muscle one after the other in immediate repetition. When the fibrillary contractions are very severe, the disease-process, at least in my experience, is rapid, the wasting of the muscle notably increasing from day to day under observation. In the slowest forms of the disease, in which many months or even years are required for much destruction, the fibrillary contractions are usually sluggish.

The loss of power takes the form of a multiple paralysis,—that is, groups of muscles more or less isolated are attacked in different parts of the body. In the majority of cases the changes are somewhat symmetrical. Thus, if one region of the hand be attacked, the same region upon the other hand will be affected. This rule is not invariable, and even when the symmetry is decided it may often be noted that not precisely the same muscles

are affeeted upon the opposite side of the body. Although loss of endurance or even partial paralysis may apparently precede the loss of muscular substance, the loss of power is due to the loss of muscular substance, and not the loss of substanee to the loss of power; or, perhaps more correetly, it may be considered that both symptoms have a common basis: *i.e.*, when a spinal ganglionic cell is attaeked, the fibres of the muscles individually supplied by it suffer simultaneously in their nutrition and in their motor functions. Usually the hands are the first portions of the body to be affected, the symptoms frequently being mueh more severe in the right hand.

According to Eulenberg, the interosseous muscles are almost invariably the first to be attacked, whilst Roberts, Wachsmuth, and Friedreich state that the the ball of the thumb is usually implicated before the interosseous muscles. The first external interosseous is said to be the first to feel the influence of the disease, whilst the opponens and the adductor pollieis are more apt to suffer than the extensors, the abductors, and the flexors of the thumb. In the few cases in which I have had an opportunity to see the disease in its earliest stage the interosseous muscles were the first affected. The wasting of the muscles of the hand is usually readily perceived by the flattening of the thenar eminence and by the falling-in of the interosseous spaces. The diminished power of the interosseous muscles can usually be deteeted by noticing that when the patient attempts to abduct the index finger he separates it with less vigor from the middle finger than normally. When only one hand is attacked, the contrast of movement is often decided. Instead of attacking the hand, progressive muscular atrophy may first make itself felt in other portions of the body, and especially is this true of the deltoid muscle; but it is stated that the pectoralis major and the serratus magnus, or even the lumbar muscles, may have to bear the onset. The upper extremities, the neek, and the trunk are certainly much more frequently affected than are the legs; nevertheless, the latter do not always escape.

Owing to the loss of power in certain muscles and to the tendency to contraetures in their antagonists, the sufferers from progressive muscular atrophy are prone to assume peeuliar positions or to have extraordinary deformities. In a patient under my own

eare, the loss of power in the muscles of the neck was so great that the head perpetually fell forward, the chin almost resting upon the breast. In this ease the upper arms were much more prominently affected than were the forearms, so that whilst the man still preserved a good grip the arms were perfectly flaccid and helpless, owing to the complete paralysis of the deltoid, biceps, and trieeps.

The most charaeteristie of the deformities is that which is known as the *clawed hand* (*main en griffe*, *Klauenhand*), and which is produced by the permanent flexion of the last two phalanges of the fingers which are extended at the metacarpal joint. As was shown by Duchenne, this deformity is the result of atrophy of the internal and external interosseous muscles with the preservation of power by the extensors and flexors of the fingers. It must be remembered that this deformity is really pathognomonic of paralysis of the interosseous muscles, and is eharacteristie of progressive museular atrophy only for the reason that loss of power of the interosseous muscle is rare from other causes. If, however, from local disease of the nerves the interosseous muscles are paralyzed, the clawed hand is developed. If only one hand is clawed, the suspicion of local disease should be at once aroused. When the muscles about the shoulder-joint are paralyzed, either by sharing in the trophie changes or by the loss of the support of the museles, the ligaments suffer elongation and the joints beeome very loose, so that a subluxation readily oeeurs.

A very important symptom in the diagnosis of progressive muscular atrophy is the preservation of the electro-museular contraetility. This at first sight may appear to be at variance with the theory that the lesion in the muscle is the result of destruction of the trophic cells in the anterior eornua of the spinal eord. The explanation of the paradox, however, is simple. The destruetion of the ganglionie cells progressively involves individual cells one after the other, and, consequently, the trophie destruction of the museles compromises individual bundles of fibres one after the other. The muselc, therefore, loses power, not *en masse*, but fibre by fibre, and that portion of the muscle which rctains its funetional aetivity preserves its normal eleetrieal reaetions.

I have never seen the reaetion of degeneration demonstrated in progressive muscular atrophy, although it is affirmed by Eulen-

berg that in the later period of the disease there may be qualitative alterations in the muscular reaction,—*i.e.*, an increased reaction under anodic closure and less commonly under cathodic opening. Eulenberg states that he has never seen in progressive muscular atrophy extreme degrees of qualitative deviation from the normal reaction. The so-called diplegic contractions which Remak has affirmed to be of frequent occurrence in progressive muscular atrophy are rarely to be demonstrated. The following paragraph from Eulenberg explains the method of developing these contractions:

"Remak found that the contractions could be produced in the atrophied muscles of the arm when the positive electrode was placed in an 'irritable zone,' which extends from the first to the fifth cervical vertebra, or, still better, in the carotid fossa, or the triangle between the lower jaw and the external ear, while the negative was put below the fifth cervical vertebra. The contractions were always on the side opposite to the anode, but when the electrodes were applied in the median line they occurred on both sides. If the current was very weak they were limited to the muscles most severely affected. Remak regarded these as reflex contractions originating from the superior cervical ganglion of the sympathetic, and especially as the patient perceived a sensation behind the ball of the eye when the current was closed."

In some cases of progressive muscular atrophy the response to the faradic current appears more active than normal. This may in some instances be due to wasting of the muscle, enabling the current more rapidly and thoroughly to reach the portion of the muscle left; but it would seem that there is sometimes a heightened irritability of the muscular fibres which have not suffered degeneration, and I have thought this was especially present when the fibrillary contractions were very severe. Again, in those cases in which the muscle as it wastes is replaced by fatty tissue, the electro-muscular contractility may appear to be below normal on account of the resistance which the fatty matter offers to the faradic current.

The course of a true idiopathic progressive muscular atrophy is usually steadily progressive until the final destruction of all the affected muscles.

Glosso-Labial Palsy.—In 1861, Duchenne described, under

the name of Glosso-Labial Paralysis, a slowly progressive loss of power in the tongue, lips, palate, and muscles of the throat, which is only a form of progressive muscular atrophy. The medulla oblongata is simply the upper portion of the spinal cord, and when the nuclei within it undergo degeneration the muscles tributary to it suffer changes precisely like those produced by similar degenerations of the nuclei of the lower spinal cord. The degeneration of the nuclei of the medulla may accompany that of other spinal ganglia, when the patient suffers from progressive muscular atrophy and glosso-labial paralysis so called, or the bulbar nuclei may alone suffer when a pure glosso-labial palsy results. The symptoms in glosso-labial paralysis vary in accordance with the varying of the degenerations in the medulla. Frequently the paralysis of the tongue is the first to appear, but the tremulousness and loss of the labial articulation may precede the lingual affection.

The course of glosso-labial paralysis is entirely parallel with that of other forms of progressive muscular atrophy. Its symptoms are peculiar, on account of the connection of the affected muscles with pronunciation, and its ending comparatively rapid, because deglutition is interfered with. There are, however, the same progressive weakness, the same slow wasting, and the same fibrillary contractions in the affected muscles, with persistent retention of electro-contractility, as in other forms of progressive atrophy. The tongue is protruded more and more slowly and imperfectly, and becomes more and more tremulous. Owing to loss of control over it, the pronunciation of the lingual vowels and of the dental consonants is imperfect. The weakness of the lips shows itself by failure in articulation of the labial consonants, by the inability to whistle, by tremulousness, and, finally, by the loss of the power to contain the saliva in the mouth, which dribbles constantly. As the disease is almost always symmetrical, the mouth is not drawn to one side, but the wasting of the parts about it may be sufficient to make the orifice appear much larger than normal and to confuse the naso-labial folds. Sometimes the lips during laughter separate themselves but are incapable of spontaneously returning to their natural position, so that the patient is forced to replace them with his fingers. If the palate is markedly affected, the voice becomes nasal. Deglutition may be affected

early or late in the disorder, and, as the loss of power of swallowing is paralytic, liquids are swallowed with much difficulty, and are apt to be returned through the nose. In some cases the larynx is attacked, and the voice becomes almost inaudible, without, however, being completely lost. In those cases in which the nuclei of the respiratory nerves are implicated the respiratory muscles undergo wasting and the respiration becomes much affected. Any attempt at violent movement, or, later in the disease, even ordinary walking, may cause a severe attack of dyspnœa. At last these cyanotic crises come on spontaneously in furious paroxysms, which may occur either by day or by night. A peculiar symptom which especially characterizes this dyspnœa is a sensation of excessive fulness of the chest, which is probably produced by the feebleness of the muscles preventing them from thoroughly emptying the lungs. In some cases the nuclei of the cardiac nerves appear to be attacked, and cardiac crises become violent and alarming. These are especially apt to be present in those cases in which the respiration is affected, but may occur without the respiratory muscles suffering. The pulse in the cardiac crises is very feeble, irregular, intermittent, and at last may be imperceptible. The face is exceedingly pale and anxious, and there is habitually an intense terror, with a sense of impending death. The ocular muscles may be affected in glosso-labial paralysis, although they usually escape.

The "ophthalmoplegia externa" of Hutchinson is in some cases the expression of a progressive muscular atrophy. (See chapter on Special Senses.)

Although glosso-labial paralysis occurs with sufficient frequency to be recognized as a clinical group, it must be remembered that all sorts of irregularities exist in the method of attack, that certain muscles about the face may be affected simultaneously with distant muscles in the body, and that the disease is only one of the many forms of progressive muscular atrophy.

Progressive Facial Hemiatrophy.—Atrophy of one side of the face requires mention here, because it may possibly be a trophic lesion due to some central disease allied to poliomyelitis, although at present this cannot be considered as established. The disease usually appears first as a whitish spot in the skin, which soon becomes brownish. Sometimes there are several of these

spots, which finally coalesce. In a little while the skin becomes thinner, so that a depression is produced. As the change widens, the folds of the skin fade, and the surface grows smooth and parchment-like. Then the subcutaneous cellular tissue atrophies. The muscular tissue yields slowly. The bones, and even the cartilages, especially of the nose, finally waste. The teeth may fall out; and the tongue and palate have, in some cases, participated in the changes. The hair becomes white or is shed.

In scleroderma and leprosy alterations of the nerve-trunks have been found, and the lesions of both affections are by some writers considered to be trophic: at present this, however, seems scarcely probable.

TROPHIC CHANGES IN THE NERVOUS SYSTEM.

The conducting nerve-fibres, both in the brain and in the spinal cord, and in the nerves themselves, appear to have their nutrition regulated by certain ganglionic cells with which they are connected, so that when isolated from such cells they undergo degeneration. These trophic changes in the nervous system are usually so hidden from any possible external examination that they can be known during life only by their secondary effects, of which muscular contracture is the only one definitely established. These contractures have already been sufficiently discussed (see page 167), and any further remarks upon the matter of trophic alterations of the nervous system may seem out of place in a work upon symptomatology. Nevertheless, I shall point out, in a few words, the laws which govern the trophic changes of nerve-fibres, and the directions in which such changes travel.

The trophic cells which dominate the fibres of the pyramidal fasciculi are situated in the cerebral cortex, so that when the pyramidal fasciculus is broken anywhere, either in the brain, in the peduncles, in the medulla oblongata, or in the spinal cord, degeneration always begins upon the lower or distal side of the injury, and travels downward until it reaches the ganglionic cells in the spinal cord. These cells are new trophic centres governing the motor nerves. We have no knowledge that the descending degeneration ever passes over from the fibres of the pyramidal tract to the trophic centres in the cord. Hence it is that in cerebral palsies the muscles preserve their integrity for

such a length of time. Whenever the motor fibres passing from the ganglionic spinal cells outward are injured, whether such injury be situated in the cord, in the nerve-root, or in the trunk, degeneration always begins in the lower or peripheral segment of the nerve, and travels downward until the peripheral filaments of the nerve are involved. In other words, in the *motor* system the trophic influence rises from higher nerve-centres, and degeneration therefore travels downward and outward.

In the *sensory* system the trophic influence originates in the peripheral or lower ganglionic masses, so that the course of secondary degeneration is from below upward. Thus, as was originally discovered by Waller, if the nerve-roots are divided above the ganglia of the posterior roots, whilst the motor root degenerates below the section, the sensory root degenerates above the section. Again, if the spinal cord be divided, in the animal by the knife, or in man by a disease (such as the transverse myelitis which often accompanies Pott's disease), below the point of section the antero-lateral columns which contain the descending motor fibres of the pyramidal tract undergo degeneration, which progresses downward. The descending degeneration travels more rapidly along the lateral columns, so that at a certain height in the cord it can be noted that they have undergone change, whilst the anterior fibres are as yet intact. Above the point of section the antero-lateral columns remain normal, but the posterior columns are altered through their whole extent. The lesion travels more rapidly along that band of the fibres which lies next to the posterior fissure and is known as the columns of Goll, so that at a certain height these fibres are diseased, whilst the posterior root-zone is unaffected. When the original lesion is situated in the dorsal region, the ascending degeneration is not confined to the posterior columns, but also passes up along a fine band situated in the posterior external portion of the lateral columns, or the tract to which has been given the name of direct cerebellar fasciculus.

In cases of inflammations of nerves the neuritis frequently travels upward along the trunk. There is, however, no reason for supposing that trophic influence has anything to do with this progression. It is simply a propagation of the inflammation by continuity, in accordance with a well-known general law.

CHAPTER VI.

SENSORY PARALYSIS.

In the present volume the term anæsthesia is used as equivalent to paralysis of sensation. Like motor palsy, it may be complete or incomplete; but, since sensation is, unlike motion, a complex function, sensory palsies vary not only in degree but also in kind and in position. An anæsthesia may affect the surface of the body, when it is spoken of as cutaneous, or it may be located in mucous membranes or in muscles or in internal viscera, or, finally, it may be situated in the region of special sense. Special sense anæsthesias will be discussed in the chapter on the Special Senses, and concerning them, therefore, nothing further will be said in this place. The function of sensation is not highly developed in mucous membranes, and we are not able to distinguish the varieties of anæsthesia in these positions that are seen upon the skin.

Cutaneous sensations may be divided for clinical purposes into two groups: *first*, those which are known as general or common sensations, as pain, itching, titillation, sensual pleasure, and the feelings arising from electrical excitation; *second*, special sensations of pressure, of temperature, and of locality.*

In practical medicine cases arise in which special sensations are paralyzed, although common sensibility is preserved. It is therefore necessary to employ various tests for the recognition of the exact condition of the part to be studied. It must be remembered that the responses which we receive from these tests are made by the patients, and may be misleading, especially in hysteria and malingering. In the examination of such cases subjective symptoms cannot be relied upon, and the judgment must be formed from the objective symptoms which are beyond the control of the patient and are seen by the physician himself.

* In separating the so-called sense of locality from common sensation I have followed custom, but have never been able to convince myself that the separation is correct. It has always seemed to me that the power of separating the points of the æsthesiometer depends upon the condition of general sensibility.

Modes of Testing Sensation.—Common sensibility may be tested with any small sharp instrument, as a needle or a knife-point, or by pinching, or by means of the electric brush. The latter instrument is especially valuable, because the peripheral nerve-filaments may be intensely irritated by it without causing any structural or permanent change. In some cases of disease, although sensation is not completely abolished, its pathway is so blocked up that a much longer time is required than normal for the perception of the peripheral sensory impulse by the brain. In extreme cases this retardation of sensation is perceptible by the watch. The detection of minor degrees of it requires very delicate apparatus, and much physical training, and is of no avail in practical medicine.

The instrument used for testing the *sense of locality* is known as the *æsthesiometer*. It consists in its simplest form of a pair of ordinary compasses with blunted points. In its more refined forms it is composed of a pair of points, one of which slides upon a bar, so that the distance between the points when separated is known; or the compasses themselves may be furnished with a graduated scale. When the points of the æsthesiometer are brought into contact with the surface of the body they are felt as two points or as a single point, according as they are more or less widely separated and as the skin is more or less sensitive. The sensibility varies greatly in different parts of the skin, but, according to the results obtained by Weber and Valentin, the distance at which the points of the compass must be separated in order to be felt as two points is to some extent constant in the same region of the body in different individuals. The following may be taken as the normal scale from which any marked deviations must be regarded as pathological: the top of the tongue, 1.18 mm.; the end of the fingers, 2.25 mm.; the side of the first phalanx, 16 mm.; the back of the hand, 31 mm.; the upper arm and thigh, 37 mm. The smallest required distance is oftener less in the transverse than in the longitudinal direction of the limbs.

Although a certain degree of constancy does exist in different individuals in regard to cutaneous sensitiveness, yet the differences are so wide that, when it can be done, it is preferable to compare the affected part with the opposite side of the body rather than with any theoretic formula. Care must be taken in applying the

compasses to see that their two points are brought simultaneously in eontact with the skin, otherwise the sense of double eontact may be produeed by the alterations of time. Also the compass-points must be kept quiet and a uniformity of pressure be maintained. When the hands are the seat of the supposed loss of sensation, the use of the æsthesiometer may well be supplemented by determining whether the patient with the eyes shut can tell the differenee between a rough and a smooth objeet.

The sense of pressure is tested by laying the hand, foot, etc., upon a firm, hard surface, like that of a table, and placing graduated weights upon it. Several forms of apparatus have been invented for the purpose of testing the pressure-sense, but none of them have any material advantage over the simpler plan. In order to avoid bringing into play the museular sense, it is essential that the part on which the weights are laid be thoroughly supported. A very eonvenient method is to fill a series of ordinary shot-gun cartridge-shells with shot and wadding so as to form a regular series of weights whieh resemble one another exactly to the eye.

The power of reeognizing the *differences of temperature* may be tested by the alternate applieation of hot and cooler bodies. More or less complieated instruments have been construeted, under the name of *thermo-æsthesiometers;* but vials of water of different temperatures are sufficient for praetieal puposes. The temperature-range of most aecurate sensation lies between 27° and 30° C., then between 33° and 39° C., and lastly between 14° and 27° C. The variations above or below these limits produce simply sensations of pain. Aecording to the experiments of Nothnagel, the smallest pereeptible differences of temperature are the following: on the breast, 0.4° C.; on the back, 0.9° C.; on the back of the hand, 0.3° C.; palm of the hand, 0.4° C.; arm, 0.2° C.; back of the foot, 0.4° C.; lower extremities, from 0.5° C. to 0.6° C.; the eheek, 0.4° C. to 0.2° C.; the temples, 0.4° C. to 0.3° C. In practiee few normal individuals will recognize, I believe, differenees of temperature so small as those mentioned.

For the purposes of clinical study, anæsthesia of the surfaee of the body is best separated from anæsthesia of the mueous membranes and of other internal tissues. For these respective

anæsthesias the names Visceral and Cutaneous may be used as convenient, although not absolutely correct.

VISCERAL ANÆSTHESIAS.

The important visceral anæsthesias met with in practice are those of the throat, rectum, bladder, and vagina. It is probable that certain obscure affections of the internal viscera may be connected with paralysis of their sensory apparatus, but of such diseases or such affections we have at present no definite knowledge. Like cutaneous anæsthesia, these deeper-seated losses of sensation may be either of hysterical or of organic origin. The distinctions between hysterical and organic anæsthesia will be fully developed in the section on Cutaneous Anæsthesia. For the present, I shall merely point out the symptoms which are produced by anæsthesia of deep-seated parts, and their usual etiological relations.

Anæsthesia of Throat.—Anæsthesia limited to the throat is a rare condition, which may occur after diphtheria, or in consequence of disease of the nerve-trunks, or may be seen in other limited organic affections of the nervous system. In combination with other symptoms, buccal and pharyngeal anæsthesia is frequently present in general anæsthesia or hemianæsthesia of the hysterical or organic type. It is especially apt to be pronounced in hysterical cases. M. Chairou (*Études cliniques sur la Paralysie*, 1870) has, indeed, insisted upon the insensibility of the pharynx and of the epiglottis as almost pathognomonic of hysteria. In many cases anæsthesia of the throat produces no distinct symptoms, and is discovered only when the parts are touched. The lack of symptoms is evidently due to the fact that the loss of sensitiveness is usually either confined to one side of the throat or is not complete. A complete anæsthesia of the pharynx and upper œsophagus would suspend the reflex movements of swallowing: it probably enters largely into the difficulty of deglutition which sometimes follows diphtheria.

Rectal Anæsthesia.—Rectal anæsthesia may be due to hysteria, to wide-spread degenerations of the brain-cortex, to myelitis, or to locomotor ataxia: when it is complete the desire for defecation does not exist, and the fæces may be retained in the rectum until there is, as in the incontinence of urinary retention, an overflow,

which is manifested by a perpetual discharge of small masses of fæces. If the fæces are hard from lack of secretion, the rectum becomes distended with an enormous stony mass. If the discharges are moderately soft, the physician is usually informed that the patient suffers from diarrhœa. In some of these cases not only the rectum but also the anus and its surroundings are distinctly anæsthetic. Sensibility may, however, be perfectly preserved in the skin and mucous membranes upon the verge of the anus, although no amount of stretching of the sphincter or intestine hurts the patient (Mitchell). An anæsthesia of one side of the rectum probably often occurs in cases of organic, and perhaps also of hysterical, hemianæsthesia, but is not discovered because the sensitive side of the rectum is sufficiently alive to perform all the necessary functions.

Vaginal Anæsthesia.—Vaginal anæsthesia is a not rare hysterical disorder. It is usually associated with a loss of sensibility in all the organs of generation, and complete loss of sexual desire, sometimes even absolute repugnance to sexual intercourse. Except in married women, it generally escapes notice.

Bladder Anæsthesia.—Anæsthesia of the bladder gives rise to retention of urine as its chief symptom. It is especially in this form of urinary retention that a perpetual overflow—*i.e.*, incontinence—is liable to mislead the unwary into believing that the bladder is sufficiently emptied. This danger is much increased by the lack of desire for urination, a symptom which is almost diagnostic of the condition. Sensory paralysis of the bladder occurs in hysteria, and is an occasional symptom of locomotor ataxia. When it is due to posterior sclerosis of the cord it is associated with genito-urinary pain-crises, which often serve to distinguish it from the hysterical disorder.

CUTANEOUS ANÆSTHESIA.

Cutaneous anæsthesia* is in its location parallel with motor palsy. It may affect the whole or the greater portion of the surface of the body, constituting a general anæsthesia. It may be

* Hereafter in this book the term anæsthesia will be used as signifying cutaneous anæsthesia.

limited to one lateral half of the body, constituting the so-called hemianæsthesia. It may be confined to one extremity, when it is known as monoanæsthesia; and precisely as two monoplegias may coexist, so we may have a double monoanæsthesia, which may simulate hemianæsthesia, or, existing upon the opposite sides of the body, may produce a crossed sensory paralysis. Anæsthesia may affect the lower half of the person, constituting a paranæsthesia. It may affect only the territory under the domination of one nerve, or of one group of nerves, constituting a local anæsthesia.

For the purposes of study, cases of anæsthesia are best arranged in certain etiological groups,—namely, hysterical anæsthesia, psychic anæsthesia, organic anæsthesia, and toxæmic anæsthesia. The last two groups might without violence to nature be considered together, but the changes in the sensory nerves which occur in toxæmic anæsthesia may not be sufficiently gross to be recognized by the microscope; moreover, the distinction between functional and organic diseases is an arbitrary one, and it is clinically more convenient to study toxæmic anæsthesias as a single group.

Psychic anæsthesia—*i.e.*, anæsthesia connected with psychosis—is no doubt dependent upon changes in the brain-cortex, which may or may not be sufficiently gross to be recognized, but, for reasons similar to those just adduced, I prefer to consider it as separate from organic anæsthesia. There is no positive relation between the etiology of an anæsthesia and its location. An hysterical anæsthesia or an organic anæsthesia may take upon itself any one of the forms based upon the distribution of the palsy and constitute a monoanæsthesia or hemianæsthesia, etc., as the case may be. A crossed anæsthesia is, however, very rarely organic, while a toxæmic anæsthesia usually is wide-spread in its distribution, although it may affect local areas which are more or less numerous and more or less widely separated.

HYSTERICAL ANÆSTHESIA.

Hysterical anæsthesia may be confined to a limited area, but is usually wide-spread: frequently it is strictly confined for the time being to one-half of the body, either as a hemianæsthesia or as a paranæsthesia; not rarely it is irregular in its distribution, and it may exist upon opposite sides of the body. It especially

affects the skin, but may make itself manifest in the deepest structures. It may be complete or incomplete. In a case reported by Briquet, a young girl had complete sensory paralysis of the skin and the muscles; the hearing and vision of the left side were gone, and the senses of taste and smell entirely lost. Her insensibility was so complete that, after the eyes were bandaged, she had no perception of being lifted from the carriage to the bed. In many cases of hysterical anæsthesia the loss of sensibility is limited to small portions of the trunk, and in a case reported by Leroy it was confined to the conjunctiva and cornea of one eye. M. Fournier has reported as occurring in nervous syphilis a localized anæsthesia of the skin, of the hands, and of the mammary region, which was almost certainly of hysterical origin. Not rarely the hysterical anæsthesia occupies the lower half, or the lower two-thirds, or a greater or less fractional part, of the body. It is rare in the face, but certainly does occur there: it is exceedingly uncommon for it to implicate the whole face.

Hysterical anæsthesia may be complete or incomplete. In it especially occurs *thermo-anæsthesia*,—*i.e.*, that condition in which the power of distinguishing between heat and cold is lost, although general sensibility is preserved. *Analgesia*, or loss of the pain-sense, existing by itself is also almost invariably hysterical. Thermo-anæsthesia and analgesia may coexist in hysterical subjects, but in the majority of cases the paralysis affects all the sensory functions.

A phenomenon which is usually present in marked hysterical anæsthesia is the so-called *ischæmia*. In this condition the surface is pale, and the prick of a needle or even an extensive superficial incised wound does not produce bleeding. Anæsthetic isehæmia appears to be specially pronounced in the violent epidemic forms of hysteria, such as occurred in the *convulsionnaires* of the Middle Ages: hence the miracle that superficial wounds were not followed by loss of blood.

When, under the influence of local applications of metals, mustard plasters, or other active or indifferent substances (see page 260), sensation returns temporarily in a case of hysterical hemianæsthesia, the ischæmia disappears and the needle-prick bleeds. Ischæmia has been held to be characteristic of hysterical hemianæsthesia, but it has been observed by Dr. S. Weir Mitchell in

cerebral hemianæsthesia, and also in the complete loss of sensation which follows nerve-section. Thus, in a case in which the sciatic and crural nerves had been divided, repeated punctures with a very large needle were not followed by any blood: as the needle was withdrawn, a small snow-white ring, slightly raised, formed around the orifice and seemed to close it.

Hysterical Hemianæsthesia.—Of the varieties of hysterical anæsthesia, hemianæsthesia is the most important, because of its frequency and of its close simulation of the organic affection. In its full extent it occupies one side of the body, and affects the special senses, causing deafness, loss of smell and of taste, and disturbances of vision. The latter may take the form of more or less complete amblyopia, but usually there is a concentric narrowing of the field of vision and a peculiar loss of color-sense, to which the name of *achromatopsia* has been given by Galezowski. In some cases the power of seeing the colors is entirely lost, so that all objects appear of a uniform sepia-tint. When the achromatopsia is not complete, the colors disappear in a constant order. The first color that an hysterical person ceases to see is violet; usually, but not always, blue is lost before red, the intermediate tints fading out in regular succession. The loss of sensibility in hysterical hemianæsthesia is distinctly limited by a line drawn through the centre of the body.

Metallotherapy.—In 1849, Dr. Burk discovered that in hysterical anæsthesia it was possible, by the application of metals to the surface of the body, to recall sensibility, and in 1851 he presented an inaugural thesis upon the subject to the Faculty of Paris. It was not, however, until 1876 that he succeeded in attracting the general professional attention of France to the matter. In that year, in answer to his importunities, the Société de Biologie of Paris appointed a commission to examine into the accuracy of his alleged facts. The report of this commission (Paris, 1879) confirmed the statements of Dr. Burk, and also extended our knowledge of the subject. It was found that different individuals have different relations with metallic substances, some cases being affected by zinc, others by iron, others by gold, copper, etc. In exceptional instances the hysterical person has relations with two or even more metals. When a small disk of the appropriate metal is bound over the anæsthetic surface of an hysterical

subject, after from ten to twenty minutes a sensation of warmth is developed beneath the disk, and a distinct reddish color appears. At this time the prick of a needle is distinctly felt, even painfully so, not only at the spot over which the plate has been applied, but also in a more or less extended zone around it. In some cases the sensibility returns only in the immediate vicinity of the application; in others the whole arm, or more rarely the whole side of the body, becomes sensitive. With the return of sensitiveness there is a disappearance of the ischæmia, and if motor palsy has existed there is also an increase of the motor power as measured by the dynamometer. In most cases of hysterical anæsthesia there is a distinct coldness of the surface, or indeed of the whole arm, and with the disappearance of the palsy of sensation and of motion there is an increase in the temperature. Thus, in a case of right-sided hysterical anæsthesia and amyosthenia, the thermometer held in the right hand stood at 36° C., in the left at 34.5° C. (Dr. Dumontpallier, *La Métalloscopie*, Paris, 1880), but after the application of the metal the temperature of the left hand was higher than that of the right. In many instances not only is the sensibility of the skin restored, but at the same time the special senses gradually become nearly normal, although in other cases it is necessary in order to affect the special senses that the metallic plates should be in the neighborhood of the orbit or in the temporal region. When achromatopsia is relieved, blue is usually the first color to return, or more rarely red. Some minutes after this, yellow is perceived, then green, and at last violet (Dr. Aigre, *La Métalloscopie*, Paris, 1879, p. 23). As seems to have been first discovered by M. Gellé, at the time of the disappearance of the anæsthesia under the influence of the metal the loss of sensibility appears in a corresponding position upon the unparalyzed side, and is accompanied by a fall of the local temperature. In a few cases severe pains have developed during the application of the metals. According to the experience of the French commission, which seems to be identical with that of subsequent observers, the effect of the application is usually in hysteria at first temporary, and lasts from a few minutes to some hours.

Dr. Burk, in his communication to the Société de Biologie, stated that if the metal which had been found temporarily to

affect sensation in a person suffering from hysterical anæsthesia were given to such patient in continuous doses, all symptoms of hysteria would after a time permanently disappear. The commission confirmed, in a measure, this statement: in sundry cases they found under such administration that menstruation became regular, digestion improved, and the muscular force and sensibility returned. They further, however, made the extraordinary discovery that if a piece of the metal were bound down on the skin of the person who had recovered, a return both of anæsthesia and of motor palsy took place in from twenty to forty minutes.

It having been suggested that the metal upon the skin acts by induction of feeble galvanic currents, the French commission found that the application of most metals to the surface of the human body gives rise to an electric current sufficiently powerful to be measured, that these currents vary in power with different metals, and that electrical currents of power equal to that of those produced by the appropriate metals applied to the anæsthetic surface brought about a return of sensibility. The observations of M. Luys showed that the application of the appropriate metals was also able to reduce hysterical hyperæsthesia to the norm.

That the phenomena of the so-called metallo-therapy as I have summarized them may frequently be obtained, in more or less completeness, is shown by the confirmation of the report of the French commission not only by a number of French observers, but also in England by Dr. A. Hughes Bennett (*Brain*, vol. i. part 3; *Brit. Med. Journ.*, Nov. 25, 1878), in Italy by Buccola and Sepilli (*Lond. Med. Record*, vol. ix.), and in Germany by Dr. F. Gratz (*ibid.*) and various other observers. It is, however, certain that, at least in this country, they are exceptional. In an elaborate series of observations made in the wards of the Philadelphia Hospital by my colleague, Dr. C. K. Mills, the transfer of sensibility was obtained in only a very few cases, whilst Dr. S. Weir Mitchell affirms as the result of his great experience that neither he nor any of his assistants have ever been able to bring about anæsthesia of the sound side, although they have very frequently obtained temporary returns of sensibility by the application of various substances, especially by mustard plasters, and even more pronouncedly by freezing the skin with rhigolene. It was at first believed that the production of sensi-

bility by æsthesiogenetic agents is proof of the hysterical nature of an anæsthesia; but in the course of his early observations upon the subject M. Charcot found that even in organic hemianæsthesia the application of the plates of metal was followed in twenty or thirty minutes by a return of the normal sensibility and of the special senses. These observations have been confirmed by several French observers.

It is also asserted that if powerful magnets be used instead of metal plates in cases of hysterical or organic hemianæsthesia with contractures and motor palsy, there will be relief not only of the paralysis of sensibility but also of the disturbances of motility. Thus, M. Laboulbène reports a case (*Gazette des Hôpitaux*) of a man, sixty-seven years of age, suffering from organic left hemiplegia and complete hemianæsthesia, in whom the application of a strong magnet was followed by the reappearance, first in the arm and afterwards in the leg, of the normal sensibility, and by a marked increase of the motor power in the hand as tested by the dynamometer. It is, however, to be noted that, so far as my examinations of the records go, there has not as yet been reported a case of organic hemianæsthesia in which any transfer of anæsthesia has been noted.

The explanation of the facts of metallo-therapy is a matter of difficulty, and no theory has as yet been offered which is satisfactory. That the phenomena are not the result of the action of a feeble electric current upon the peripheral nerves seems to be shown by their having been produced by metals, such as platinum, which are practically non-oxidizable, and by absolutely inert substances, such as disks of wood, and even, as in the case reported by Bennett (*loc. cit.*), by the application of a handkerchief. The theory adopted by most English writers, that they are the result of expectant attention,—*i.e.*, that they are the result of the patient's believing that the phenomena are about to happen,—is asserted to be disproved by the fact that in many cases the patient did not know what was to happen. The so-called molecular theory, which teaches that there is some mysterious molecular influence produced by the applied plate on the peripheral nerve-filaments, amounts to nothing more than words.

Diagnosis of Hysterical Anæsthesia.—Only in rare cases is there any difficulty in distinguishing between an hysterical and an

organic anæsthesia. Hemianæsthesia occurring in a woman is usually hysterical; in man it is commonly organic. There is a peculiar atmosphere surrounding the hysterical person which to the experienced physician reveals the nature of the case, even when there are no distinct symptoms of hysteria other than the disturbances of sensibility. Almost invariably, however, a history can be elicited of past convulsive seizures or of shifting paralysis, of globus hystericus, of caprices of temper or disposition, or of other hysterical manifestations. Further, in organic cases the form of the palsy, the disturbances of intellection, and the history of the case generally strongly indicate the existence of organic disease. In the great majority of cases of hysterical hemianæsthesia the patient does not know of the existence of the condition. If a motor and a sensory paralysis coexist, they are as likely as not upon opposite sides of the body in hysterical anæsthesia, whilst in cerebral hemianæsthesia they are of necessity upon the same side, unless, indeed, there be two distinct lesions in opposite hemispheres. According to Briquet, the hemianæsthesia of hysteria is in seventy per cent. of the cases upon the left side.

The recognition of hysterical hemianæsthesia is further facilitated by attention to the following considerations:

First. The organic anæsthesia is fixed, and does not vary from day to day in its limits, whereas in the hysterical disorder very often the locality of the sensory palsy varies markedly from day to day; and even when this does not occur, the exact limits of sensation can be noted to shrink and increase perpetually.

Secondly. In hysterical hemianæsthesia there are usually spots located within the general anæsthetic region in which there is hyperæsthesia or normal sensation. The school of Charcot has especially directed attention to the almost universal presence of hyperæsthesia of the ovary upon the affected side. In America this ovarian hyperæsthesia can very rarely be demonstrated, but, as has been elaborately detailed by Mitchell, there is frequently a region in the groin in which hyperæsthesia exists, although the ovary may not be affected. This territory reaches from the line of the groin upward, sometimes as far as the navel. The sensitiveness may be limited to the skin, or may be felt only upon deep pressure, or may be both superficial and deep-seated. Its

presenee has been noted by Mitchell in cases in which the ovary had been removed by the surgeon.

It is probable that spots or tracts of sensitiveness frequently oeeur in the midst of the anæsthetic region and are overlooked. M. Fère (*Archives de Neurologie*, 1882) found such a sensitive spot, the size of the hand, between the dorsal and lumbar regions, and Dr. Mitehell has called espeeial attention to the frequeney with whieh the anæsthesia is wanting in a limited vertieal spaee, from one to two inehes wide, stretching from the lower eervical region to some position in the dorsal region.

Psychic Anæsthesia.—In 1883 (*Neurol. Centralb.*, xxiii.), Dr. R. Thomsen announced that eutaneous and sensory anæsthesias often exist in epileptics, and in connection with H. Oppenheim (*Arch. für Psychiatrie*, xv. 558) he published, in 1884, an elaborate paper upon the subjeet. An examination of ninety-four cases of epilepsy showed that no sensory disturbance follows the ordinary motor epileptic attaek, and that there is no permanent alteration of sensibility, except in the case of old epileptics, who present other more or less distinct symptoms of permanent functional or organie degradation of the cerebral cortex. Temporary anæsthesia was found to oeeur after attaeks under three eircumstances:

First, when the convulsion was followed by post-epileptic delirium with hallueinations.

Seeondly, when the attack was followed by violent emotional or psychieal disturbanee without delirium.

Thirdly, when the motor epileptic convulsion was replaced by an abortive attaek more or less violently affecting the mental or emotional sphere.

It would appear from these researehes that when the epileptie diseharge does not ehiefly or solely affect the motor sphere, but spreads itself through the upper brain-eentres and eauses intelleetual or emotional disturbanee, it is very apt at the same time to exert its influence upon those portions of the cortex which are conneeted with sensation.

The absence of sensibility in many eases of insanity is notorious, but, for obvious reasons, in the insane it is exceedingly difficult to determine the exact limits or extent of the loss of sensitiveness. Espeeially is this true of acute dementia, melancholy, and aeute mania.

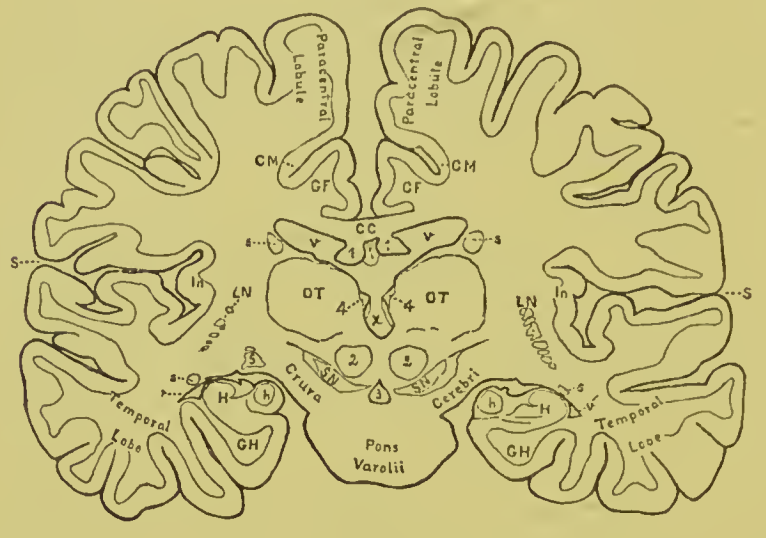
ORGANIC ANÆSTHESIAS.

Description of the Sensory Pathways.—Before discussing anæsthesias of organic origin it seems proper to point out very briefly the pathway which sensory impulses follow in going from the periphery to the respective centres of the brain-cortex. From the surfaces of the body pass the sensory nerve-fibres, which enter the spinal cord through the so-called posterior root-zones and go to the gray matter.

The decussation of the spinal fibres has been much discussed. It appears to be the general belief of physiologists that the immediate sensory decussation is not complete, although the major portion of the fibres do cross over directly after entering the spinal cord: in accordance with this view, a sensory impulse passing upward from the foot crosses in large part to the lumbar region of the cord, but to some extent continues upward without decussation to the dorsal or possibly even as far as the cervical spinal cord. The exact upward pathway of sensory impulses after decussation cannot be considered as finally determined; but the physiological evidence which we have indicates that, whilst motor impulses descend along the antero-lateral columns of the cord, sensory impulses pass upward through the posterior columns and the gray matter. Many physiologists teach that the sensations of general sensibility and the afferent impulses which give rise to reflex acts are transmitted by the gray matter in all directions, whilst tactile sensations travel exclusively by the posterior columns. This, however, does not seem to be fully proved: the facts of disease simply warrant the conclusion that afferent impulses of all characters find their way either through the posterior columns or through the central gray matter, and that an interrupting lesion of these regions is followed by loss of sensation of all characters in the parts below the lesion. The sensory pathways through the medulla correspond in position with those of other portions of the spinal cord: in the pons they occupy the outer nerve-bundles; in the peduncles they lie in the so-called tegmentum,—*i.e.*, upon the posterior or superior portions, separated more or less distinctly from the crusta, or motor pathway, by the ganglionic mass known as the locus niger. As was first pointed out by Meynert, after leaving the peduncles the sensory

fibres pass upward and backward and form the posterior third of the so-called internal capsule, in immediate relation with the

Fig. 9.



posterior and external aspect of the optic thalamus on the one hand and with the posterior part of the lenticular nucleus on the other (see Figs. 9 and 10): at this position the ascending sensory spinal fibres are joined by other fibres coming upward through the corpora geniculata and the optic thalami from the optic tract. Owing to the conjunction of all the sensory fibres, a lesion at this position produces a complete anæsthesia of special and general sensation upon the opposite side of the body. From the internal capsule the sensory fibres radiate in the so-called corona radiata. Their exact termination in the cortex remains at present a matter of doubt.

ORGANIC GENERAL ANÆSTHESIA.

A general anæsthesia of organic origin is usually due to a wide-spread degeneration of the brain-cortex, or to a double lesion of the cortex, or to a lesion occupying the posterior third of the internal capsules of the two hemispheres, or to a wide-spread peripheral neuritis. The diagnosis of the site of the lesion must be made from the concomitant symptoms. If there be dementia pointing to profound degeneration of the cerebral cortex, the diagnosis of such lesion is made out; if there be pain and marked tenderness over the nerve-trunks, peripheral neuritis is indicated.

A spinal general anæsthesia is theoretically possible, but in order for it to involve the face the lesion would have to be situated in the medulla oblongata. If in any case of general anæsthesia there were a history of slow progressive development, with symptoms of spinal implication and no neural tenderness, the probable diagnosis would be an ascending chronic lesion either of the posterior columns of the spinal cord or of the central gray matter.

Gouty Anæsthesia.—Dr. J. A. Ormerod has called attention to a variety of general anæsthesia in which the patient on awakening has a feeling of numbness, deadness, pins and needles in the hands and arms, and sometimes also in the legs. There is also loss of power, and occasionally the hands are so far paralyzed that the patient is unable to hold anything. Sometimes the symptoms pass off in the course of a few hours, in other cases they leave more or less permanent disablement, and even the temporary attacks are prone to recur. In some of these cases the victim is gouty, and with the anæsthesia are associated severe pain and stiffness. Such cases must be looked upon as probable instances of gouty multiple neuritis. In others of the cases reported by Dr. Ormerod the affection was plainly hysterical.

ORGANIC HEMIANÆSTHESIA.

Organic hemianæsthesias for clinical purposes are best divided into—first, those in which the special senses are not involved at all; secondly, those in which the senses of taste and smell alone are implicated; thirdly, those in which all the special senses are affected.

Organic Hemianæsthesia without Involvement of Special Senses.

Organic hemianæsthesia without involvement of the special senses may, theoretically, be due to a spinal lesion situated very high up; to disease of the medulla oblongata; to lesion of the pons; to structural changes in the peduncle, the internal capsules, or the cortex of the occipital lobes.

Spinal Hemianæsthesia.—In spinal unilateral loss of sensation the loss of sensation is upon the side opposite to the lesion, whilst any motor paralysis which is present is upon the side of the lesion. If the lesion be of such character (a gummatous

tumor springing from the membranes, for example) as to involve nerve-roots, an upper zone of anæsthesia may exist upon the side of the lesion along the upper margin of the motor paralysis. The motor and sensory disturbances never extend above the spinal lesion, and as the latter, if above the origin of the respiratory nerves, would of necessity produce fatal respiratory paralysis, spinal hemianæsthesia never involves the face or the neck, and very rarely the arms.

A gummatous tumor or a transverse myelitis, whilst affecting chiefly one side of the cord, may at the same time influence to some extent the other side. The result would be complete motor palsy on the side of the lesion and anæsthesia on the side opposite to the lesion, with partial loss of power on the side opposite to the lesion and partial loss of sensibility on the side of the lesion,—all these symptoms being present only in those portions of the body which are below the spinal lesion. To make this more clear, suppose that the tumor existed upon the left side of the upper dorsal cord: then there would be complete loss of sensibility in the right leg, and partial loss in the left leg, whilst motion would be entirely lost in the left leg, and partially in the right leg.

Bulbar Hemianæsthesia.—Acute lesions of the medulla produce such stormy symptoms and so rapidly fatal a paralysis that sensory disturbances are very rarely noted: when they exist, they almost invariably take the form of a partial general anæsthesia.

Hemianæsthesia from Disease of Pons.—A hemianæsthesia without involvement of the special senses may be due to disease of the pons, when it is almost always associated with hemiplegia. According to the collection of cases made by Couty (*Gazette Hebdom.*, 1877, vol. xiv.) and by Nothnagel, small lesions situated in the middle portion of the pons produce no anæsthesia of the extremities, but only motor paralysis, whilst anæsthesia results when the lesion is situated more to one side or near the floor of the fourth ventricle. Our pathological material does not seem sufficient to warrant a positive formulation of the apparent law, but indicates that destruction of the outer bundles of the pons is necessary for the production of anæsthesia.

Owing to the high origin of the trigeminus nerve, a crossed

sensory paralysis may be produced by a lesion of the pons; that is, the left face and the right side of the body may be anæsthetic, or *vice versa*. Strictly unilateral anæsthesia may, however, result from a disease of the pons. The following scheme, taken from the paper of Dr. Sigerson (*Dublin Med. Journ.*, vol. lxv., 1878), reports the different forms of paralysis which may be produced by disease of the pons, and suggests names for such varieties:

1. SIMPLE ALTERNATE PARALYSIS (MOTOR OR SENSORY).

Bend-dexter.—Right face and left extremities.
Bend-sinister.—Left face and right extremities.

2. COINCIDENT ALTERNATE PARALYSIS.

Sensory and motor paralysis of same regions.

3. DOUBLE ALTERNATE PARALYSIS.

Complete.

X-shaped Paralysis.—Both sides of face and extremities of both sides of body affected.

Incomplete.

V-shaped Paralysis.—Both sides of face affected.

Y-shaped Paralysis.—Both sides of face and extremities of one side of body affected.

Lambda-shaped Paralysis.—One side of face and extremities of both sides of body more or less affected.

Hemianæsthesia from Lesion of Peduncle.—A general hemianæsthesia without involvement of the special senses may result from a lesion of the peduncle. I know of no case in literature in which sensory without motor paralysis has been proved by an autopsy to have been due to a lesion of the peduncle, but in a case reported by M. Mayor (quoted by Nothnagel) a small focal disease was found in the inner half of the peduncle, and during life motor palsy had existed without sensory disturbance. This would indicate that the sensory fibres run through the outer or superior portion of the peduncle. In all reported cases of lesion of the peduncle the hemianæsthesia and hemiplegia have both been on the side opposite to the lesion, whilst the oculo-motor

paralysis, when it has existed, has been upon the same side as the lesion, and therefore is crossed with the hemianæsthesia and the hemiplegia.*

Fig. 10.



Cerebral Hemianæsthesia.—A unilateral sensory paralysis not implicating the special senses may be due to a lesion in the brain, provided such lesion be so situated as to avoid the fibres of special senses. According to Meynert, a very limited lesion in the optic thalamus near the geniculate bodies is capable of paralyzing solely the fibres connected with the special senses. A little in front of this the fibres of general sensibility are fused with those of the special senses. More in front the fibres connected with general sensibility exist by themselves, and consequently a minute lesion at such point would produce a hemianæsthesia without affection of the special senses. A hemorrhage or minute tumor in the anterior portion of the optic thalamus, or in the lenticular nucleus, might therefore by pressure paralyze the fibres of general sensibility without affecting those of special

* The case reported by Dr. G. E. Paget (*Med. Times*, 1855, vol. xxxi.) has been widely quoted as exceptional. In it a tumor was found springing from the right crus, although during life there had been oculo-motor paralysis of the left side, with right hemianæsthesia and right hemiplegia. It is plain, however, that the lesion involved the left crus more than it did the right, for we are told that the "mass commenced a little posterior to the junction of the crura, and extended obliquely forward from the right *into the left crus.*" The case cannot, therefore, be considered as exceptional.

sensation, and thereby give rise to a hemianæsthesia without disorder of the senses.

The disturbance of sight which occúrs in cerebral hemianæsthesia is always an homonymous hemianopsia,—that is, a hemianopsia which affects the same side of each eye. The details and explanation of this will be given in the chapter on Special Senses. The brief formulation of the diagnostic points is, that when both nasal or both temporal fields are affected the lesion is in the optic chiasm and not within the cerebral hemispheres, but that when a temporal and a nasal field are conjointly paralyzed the region is within the cerebrum.

Hemianæsthesia involving Special Senses.

Hemianæsthesia involving some, though not all, of the special senses, although very rare, does occur. As is shown in the collection of cases made by Couty, in hemianæsthesia due to disease of the pons the senses of smell and taste may be involved without any disturbance of vision or audition. Loss of taste is to be expected in the lesion of the pons, because the gustatory fibres of the glosso-pharyngeal nerves pass through the pons; but it is difficult to explain the loss of smell, except by supposing that the pons lesion is situated so far anteriorly as to press upon the olfactory bulbs, or else that it is placed so far posteriorly as to encroach upon the immediately contiguous uncinate convolutions, in which some physiologists locate the sense of smell. Hemianæsthesia with loss of hearing and sight without implication of the senses of smell and taste probably never occurs as the result of an intracerebral lesion. I have, however, seen one case in which there was partial hemianæsthesia with epileptic attacks, Jacksonian in type, complete hemianopsia, and partial loss of hearing upon one side, with demonstrable alteration of taste and smell, all due to a band-like gumma which stretched obliquely across the anterior end of the pons, reaching as far forward as the cribriform space. The corpora quadrigemina and the optic tract were involved in the exudation, as was also the auditory nerve of one side: hence the affection of sight and hearing. One end of the tumor was much thicker and heavier than the other, and consequently there was a slight hemianæsthesia and hemiplegia. In a case of this character the basal situation of the lesion is plainly revealed during

life by the hemianopsia not being homonymous: thus, in the case just detailed, both the temporal fields were paralyzed.

Complete hemianæsthesia involving both common sensibility and the special senses not rarely coexists with hemiplegia. Under these circumstances, if the symptoms are the result of a single lesion the hemianopsia is always homonymous. The lesion must be higher up than the geniculate bodies, and must be sufficiently large to destroy or to paralyze by pressure the whole of the internal capsule, except the anterior segment, which contains cerebellar fibres: in other words, the lesion must directly or indirectly paralyze the posterior segment and the knee of the internal capsule.

I have seen several cases in which hemianopsia coexisted with hemiplegia and with absolute aphasia, but have never had an opportunity to make the autopsy of such cases. As the fibres of the facial and hypoglossal nerves are believed to run through the knee of the capsule (see Fig. 10), a lesion at such place might affect articulation; but in the cases of which I am now speaking a true amnesic aphasia existed. So far as present knowledge goes, no destruction of the internal capsule is able to produce such aphasia. The knee of the capsule is, however, about on the same level in the brain as the island of Reil, so that a large clot in the lenticular nucleus, which lies between the island of Reil and the knee of the capsule, might, by pressure, paralyze on one side Broca's convolution, and on the other side the internal capsule, thereby producing complete hemianæsthesia, hemiplegia, and aphasia. An embolus of the middle cerebral artery might also produce these symptoms by destroying a large area of the brain-cortex. When, however, symptoms in a case are complicated and, as occasionally occurs, in a measure contradictory, there is always a possibility of a double lesion.

ORGANIC PARANÆSTHESIA.

Paranæsthesia is in the vast majority of cases of spinal origin. Theoretically, it might be produced by two coexisting brain-lesions, and a multiple neuritis confined to the lower extremities would cause it. In the latter case the pain and tenderness over the nerve-trunks would reveal the nature of the affection.

For diagnostic purposes cases of paranæsthesia are to be divided into four groups:

1. Cases in which the development is abrupt.

2. Cases in which a few hours are required for the production of the symptoms.

3. Cases in which some days are necessary for the production of the symptoms.

4. Cases in which many months are required even for the partial development of the symptoms.

First. A paranæsthesia abruptly developed without much pain is characteristic of hemorrhage into the spinal cord. Under these circumstances it is complete, and affects the rectum and genito-urinary organs. A very sudden paranæsthesia is sometimes produced by embolism and consequent arrest of function and softening of a portion of the cord. It may be produced by a traumatism, such as a stab or other injury, which suddenly puts an end to the functions of the spinal cord.

Second. A paranæsthesia which has come on in the course of a few hours and has been associated with great pain is almost certainly due to rupture of a blood-vessel within the spinal membranes.

Third. A paranæsthesia which becomes nearly complete in the course of a few days usually is caused by a central myelitis or by a very rapid transverse myelitis. A more slowly developed paranæsthesia may be the result of a subacute myelitis, or of softening of the cord, or of a rapidly-developed tumor.

Fourth. Chronic sclerosis affecting the posterior columns of the cord, whether it occurs in foci or in tracts, produces below the lesion a loss of sensibility which is characterized by extreme slowness of development; by its rarely, even in its later stages, becoming complete; and by its connection with loss of knee-jerk or of co-ordination, shooting pains, or other well-known symptoms of locomotor ataxia.

ORGANIC MONOANÆSTHESIA.

Monoanæsthesia may be produced by a cortical lesion affecting centres in the occipital lobe which are connected with sensation. It may also be caused by a wide-spread traumatic or idiopathic neuritis. Thus, I have seen it as the result of concussion and subsequent inflammation of the brachial plexus, due to a curling storm-wave giving a downward stroke from above the clavicle.

ORGANIC LOCAL ANÆSTHESIA.

Local anæsthesias may be the result of narrowly-defined cortical brain-lesions, or may be produced by traumatisms or diseases of the nerves themselves. The most ordinary forms of local anæsthesias are those which accompany motor paralysis due to pressure upon nerve-trunks. A partial local anæsthesia often accompanies neuritis, and may even be associated with a true hyperæsthesia; that is, an inflamed nerve may lose its functions of special sensibility, and at the same time be excessively susceptible to the pain-reaction; so that, although the slightest touch upon the part may produce severe pain, the patient is unable accurately to locate the points of the compass or to distinguish degrees of temperature.

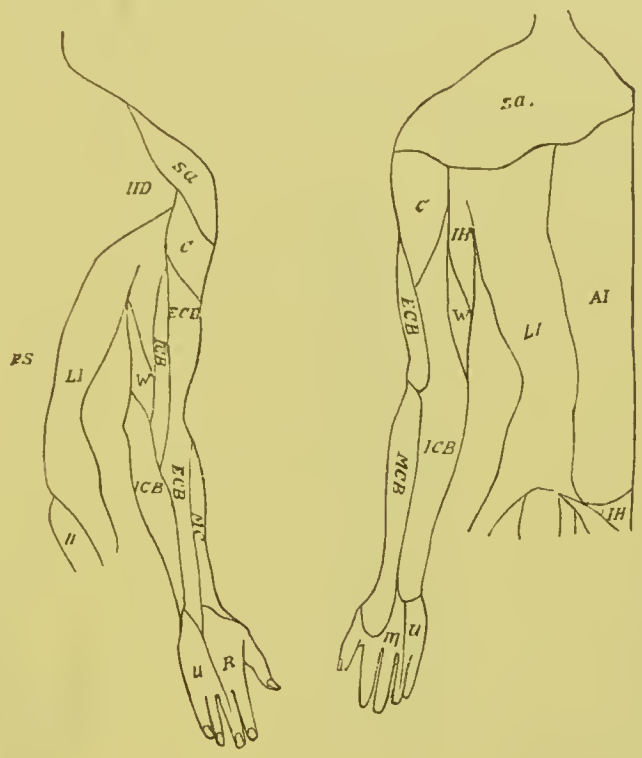
Anæsthesia produced by section of the nerve would be expected to follow the distribution of such nerve as given in standard treatises upon anatomy; but the results of clinical observation of the effects of section of nerves show that this expectation is not thoroughly realized. Thus, a principal nerve of the brachial plexus may be divided without giving rise to complete anæsthesia in any way; and when a complete anæsthesia does result, the portion of the surface so affected is very limited, and often the area of partial anæsthesia does not correspond with the generally recognized anatomical distribution of the nerve. Moreover, the division of the same nerve in different people produces different results in its relation to anæsthesia. Anæsthesia the result of nerve-section tends to become progressively less in degree and extent with the lapse of time.

I shall not enter into an elaborate discussion of this subject, but shall give a series of figures representing observations made by several observers. For details the reader is referred to the paper of Dr. James Ross (*Brain*, vol. vii. p. 50), to the work of E. Létiévant (*Traité des Sections nerveuses*, Paris, 1873), and to the articles referred to by the writers just named.

Fig. 11 represents the distribution of the cutaneous nerves as given by Flower; I H, W, I C B, respectively, representing the intercosto-humeral nerve, the nerve of Wrisberg, or small internal cutaneous nerve, and the internal cutaneous nerve, all derived from the roots of the eighth cervical and the first and second

dorsal nerves; S A, the supra-clavicular and supra-acromial branches of the cervical plexus; C, the circumflex nerve; I C B

Fig. 11.



and E C B, the internal and external cutaneous branches of the musculo-spiral nerve; M C and M C B, the cutaneous branches of the musculo-cutaneous nerve, probably derived from the fifth, sixth, and seventh cervical roots; R, the radial nerve, and U, the ulnar nerve. The distribution of the nerves in the hand given by Krause appears to be more in accord with clinical results than that which is usually adhered to by the English anatomists.

Fig. 12.



Fig. 12 represents this distribution, the letters standing for the nerves whose names they begin.

Fig. 13, after Létiévant, represents the effects of an anæsthesia which followed section of the sciatic nerve. The dark shading signifies total anæsthesia, the lighter, partial

anæsthesia: the depth of the shading represents the intensity of the loss of sensation.

Fig. 13.



Fig. 14, after James Ross, shows the interior and posterior aspects of an arm after a rupture of the brachial plexus, in which the motor fibres from the fourth cervical nerve probably escaped. The loss of sensation in the part was complete.

A section of the brachial plexus, reported by Maury and Duhring (*Amer. Jour. Med. Sci.*, ii., 1874), was followed by a loss of sensation in the forearm and a disturbance in the upper arm, indicated in Fig. 15: the interrupted line marks the boundary of the anæsthesia on the inner surface of the arm, whilst the uninterrupted line *a a* marks the boundary of the anæsthesia on the outer surface of the arm. This case would seem to show that the intercosto-humeral nerve and the descending branches of the cervical plexus supply most of the inner surface of the upper arm and a little of the outer surface.

Fig. 16, after Létiévant, represents the loss of sensation two years and a half after division of a musculo-spiral nerve by a poniard. The depth of the shading indicates the degree of sensory palsy. The area of the anæsthesia in this case is remark-

Fig. 14.



ably in contrast with that of the anæsthesia which followed in a case of Dr. S. Weir Mitchell's division of the musculo-spiral and median nerve, as represented in Fig. 17: in this figure the light shading indicates the area in which tactile sensibility was diminished, and the deep shading, that in which it was lost.

The marks $>$ $<$ indicate the appreciation of separate points. A single mark ($\vee$) indicates that the points are not felt as sepa-

Fig. 15.



Fig. 16.

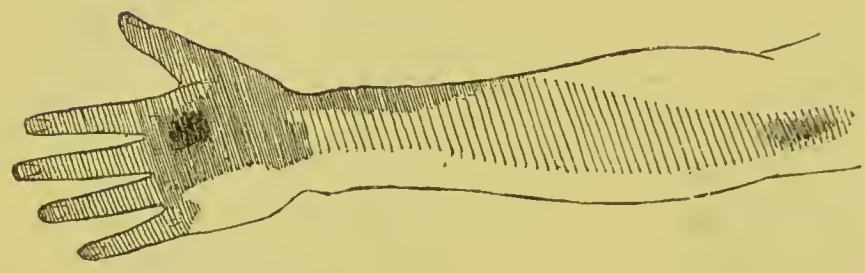
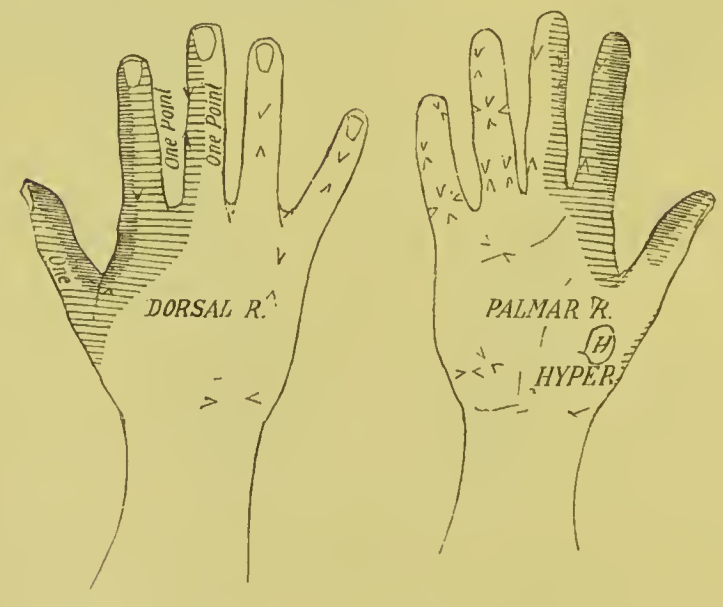


Fig. 17.



rate. The absence of the mark indicates complete loss of tactile sensibility. H indicates a small area in which there was hyperalgesia of the skin.

Fig. 18, after Létiévant, represents the distribution of anæsthesia after a complete division of the ulnar nerve at the wrist; the darkest shading indicating complete loss of sensation, the lighter shading, partial loss, in proportion to the depth of the shading.

Fig. 18.

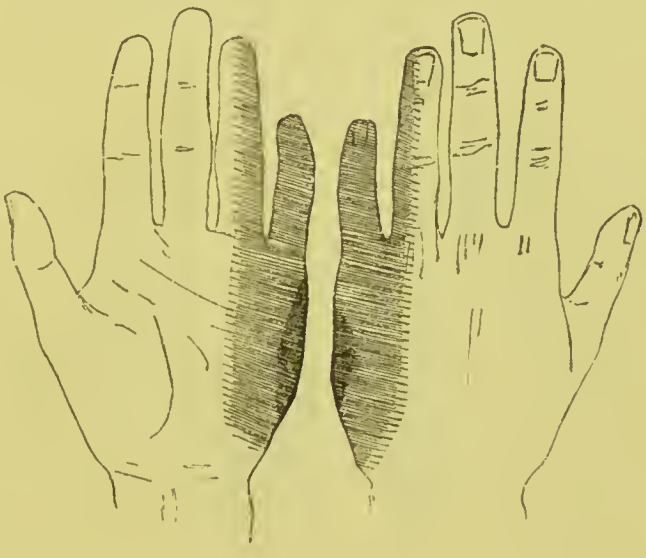
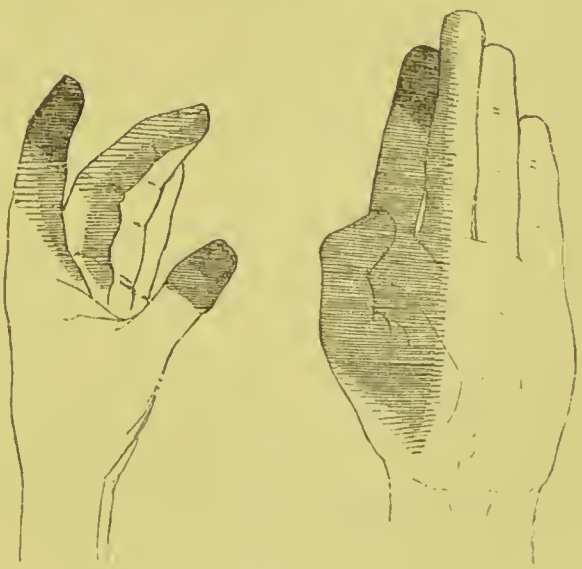


Fig. 19, after Létiévant, shows the radial border and the dorsal aspect of the hand after division of the median nerve. From

Fig. 19.



five divisions of the median nerve carefully observed, Létiévant concludes that the anæsthesia is distributed over the palmar surfaces of the thumb, index, and radial border of the middle finger,

the thenar eminence, and the radial half of the palm, and over the dorsal surfaces of the ungual and middle phalanges of the index and middle fingers, and occasionally the ungual phalanx of the thumb. The anæsthesia is only partial, except over the palmar surface of the whole of the ungual phalanx of the index finger and part of the dorsal surface of the same phalanx.

Fig. 20.

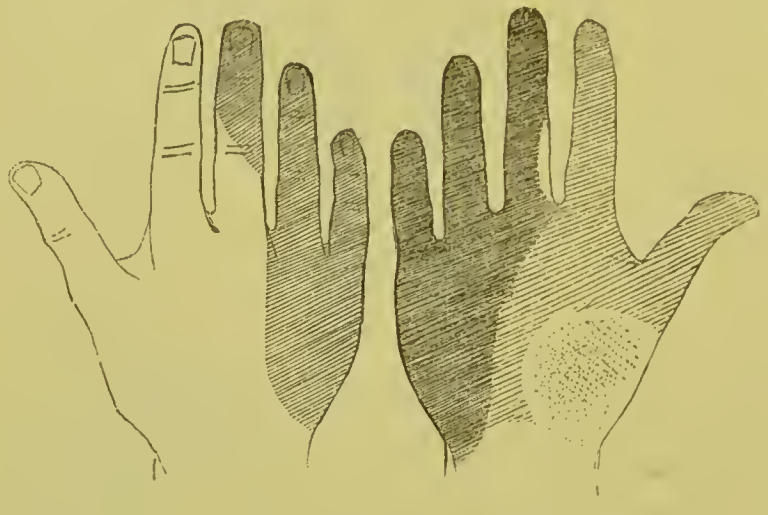


Fig. 20, after James Ross, shows the anæsthesia which followed the division of the ulnar and median nerves.

CHAPTER VII.

EXALTATIONS OF SENSIBILITY.

HYPERÆSTHESIA.

By the term hyperæsthesia, as used in this book, is meant a condition of the sensory nervous system which causes it to respond more actively than normally to irritations. This condition is closely associated with pain, but is distinct from it, since a part may be hyperæsthetic and yet not painful so long as no external irritation is present. Often a part is both painful and hyperæsthetic. Thus, an inflamed nerve suffers from pain originating within itself, and is also excessively sensitive to external irritations. Precisely as anæsthesia of special sensibilities may coexist with or may exist separately from anæsthesia of general sensibility, so may special and general hyperæsthesias exist alone or together. Hyperæsthesia or excessive functional activity of special sensibilities is, however, rare, although there are hysterical cases in which there is a positive increase of sensibility as regards the discrimination of locality and of temperature. Probably one explanation of the extreme infrequency of increase of special sensibilities is to be found in the fact that whenever a specialized nerve becomes hyperæsthetic it is very prone to respond, even to its normal stimuli, by pain rather than by extraordinary acuteness of functional activity. Thus, a hyperæsthetic eye commonly does not see more acutely than normal, but suffers intensely under the stimulus of light. There is, however, in hysteria occasionally hyperæsthesia of the special senses, in which, with or without the coexistence of the pain-reaction, there is an excessive functional power, so that the eye will see clearly in a darkened room, or the ear will hear sounds which are inaudible to the normal ear.

For the purposes of study and discussion, hyperæsthesias may be divided into Hysterical Hyperæsthesia, Psychical Hyperæsthesia, and Organic Hyperæsthesia.

HYSTERICAL HYPERÆSTHESIA.

Hysterical hyperæsthesia may follow the regional distribution which is common in anæsthesia of the same nature: usually, however, it is more irregular in its distribution, occurring in patches which may interrupt anæsthetic tracts. Concerning these interrupting tracts sufficient has already been said under the head of Anæsthesia. It seems here necessary only to point out in detail certain forms of local hysterical hyperæsthesia which are liable to be confounded with disease of other character.

Genital Hyperæsthesia.—Among the local hysterical hyperæsthesias to be here mentioned is hyperæsthesia of the genital organs. This, seen almost exclusively in the female, is rarely associated with an excess of sexual desire, but usually gives rise to severe pain during the sexual act, and often lies at the foundation of the vaginal spasm known as vaginismus.

Hysterical Breast.—An important and common form of local hyperæsthesia is the hysterical or neuralgic breast, which has frequently been mistaken for cancerous or other organic disease. The breast is often much swollen, and the pain excessive, sometimes shooting down the arm and being made worse by using the arm. The diagnosis between this condition and organic tumor of the breast can usually be made without difficulty by paying attention to the following points. In the first place, the tenderness is excessive, and is superficial, so that as much pain is produced by merely brushing or handling the breast as by hard pressure. In organic tumor the pain is proportionately increased by pressure. Again, the swelling is more diffuse in the hysterical breast, and lacks the definite limitation usually seen in organic disease. If a distinct tumor be simulated, it is commonly less persistent in form and more tender than is the organic alteration. The neurotic breast varies in size and in consistency continually, and is often enlarged and more painful at the menstrual period; the pain is often excessive, and is increased by the approach of stormy weather and by general fatigue. In almost all cases marked evidences of the neurotic or hysterical temperament exist.

A form of the neurotic breast which not rarely gives rise to alarm on the part of parents occurs at the age of puberty. When the system is expanding from childhood to womanhood the breasts

usually become swollen and tender, but if all the genital organs unfold themselves simultaneously no thought is taken about the matter. In neurotic young girls there is often, however, irregularity of sexual unfolding, so that while one breast remains as heretofore the other suddenly grows hot, and so painful and tender as materially to impede the use of the arm. I have seen a number of similar neurotic breasts occurring in boys at the age of puberty, and even attended with the secretion of a few drops of sero-lacteal fluid.

Hysterical Joints.—Hysterical patients are very liable to affections of the larger joints simulating a chronic inflammation, but due to a neurotic hyperæsthesia. Of all the larger joints the knee is the one most usually attacked. The hysterical disease of this joint is to be recognized by attention to the following considerations: *first*, the muscular rigidity or contraction can be overcome by mildly persistent efforts while the patient's mind is diverted, yields readily during natural sleep, and disappears during slight anæsthesia, or even under a full dose of chloral or opium: *secondly*, there is no rise of temperature in the joint, although the part looks red and inflamed: *thirdly*, the reaction of the contracted and apparently atrophied muscles to the faradic current is normal.

In organic disease of the knee, relaxation never takes place except in profound anæsthesia, the temperature of the surface is above normal, and the faradic reaction of the affected muscles is lost.

A mimetic disease of the hip-joint is often distinguished with some difficulty from the organic affection. It is to be recognized by noticing that the apparent tenderness of the limb as revealed by the limp varies greatly from time to time, and especially is less when the attention of the patient is diverted: it is also made worse by fatigue or nervous excitement, and hence is usually much more distinct in the evening than in the morning. Pain, which in the organic disease commonly follows the limp in the order of its development, in the hysterical disease generally precedes the limp. The muscular rigidity in the hysterical affection varies greatly, and can be readily overcome by chloral and anæsthetics, and disappears during sleep. Even if muscular atrophy exists, the normal electrical contractility is preserved. The evidences of the hys-

terieal temperament abound in this as in all other mimetie joint-affeetions, and the symptoms are mueh less severe when a great desire arises on the part of the patient to perform aets requiring exertion.

Spinal Hyperæsthesia.—An important local form of hysterical hyperæsthesia is that whieh is present in the so-ealled *spinal irritation*, or *spinal anæmia*. This condition, whieh by many writers of text-books on nervous diseases is raised to the rank of a distinct disease, is exceedingly frequent and variable in degree. In a very large proportion of neurotic young women there exists a tract somewhere in the spinal column which is espeeially tender to touch. The ordinary position is between the shoulders; the hyperæsthetie region may, however, be as low as the lower lumbar or as high as the upper cervieal vertebræ. Even in moderate eases a slight touch produces a sense of faintness, and siekness of the stomach, and I have seen eases so severe that painting with a camel's-hair brush would cause excessive agony, and, if persisted in, even general eonvulsions,—cases in whieh the laying of a hand or even the toueh of a pillow on the back would make the patient fall in the bed and lie for days apparently at the point of death, unable to turn or to speak, save in the faintest of whispers. In sueh women violent hysterical convulsions oecur oeeasionally. The symptoms are commonly intensified by fatigue, and not rarely much pain between the shoulders is complained of. This pain is increased by jarring, and by any prolonged use of the arms. There is not, to my mind, the slightest evidence of the existence of anæmia or eongestion or of any other recognizable alteration of the spinal eord in this so-ealled spinal irritation. The diagnosis is very easy: in serious disease of the vertebræ, and even in inflammation of the posterior nerve-roots, the sensitiveness is not so extreme, and usually eannot be developed exeept by firm pressure. Further, hyperæsthesia of the spinal region, unless excessively severe, is not aecompanied by spasm of the spinal museles or restriction of the movements, sueh as are seen even in incipient disease of the vertebræ. When to these facts are superadded the existenee of the hysterieal eonstitution, the absence of evidences of severe constitutional disorder, and the shifting, varying, evidently neurotic type of the tenderness itself, the eharaeter of the case becomes evident.

ORGANIC HYPERÆSTHESIA.

Organic hyperæsthesias replace organic anæsthesias when the lesion irritates rather than paralyzes: hence, theoretically at least, it is possible to have hemihyperæsthesias, parahyperæsthesias, monohyperæsthesias, etc., each corresponding to an organic anæsthesia. Clinically, however, hyperæsthesias due to disease of the nerve-centres themselves are so exceedingly rare that detailed consideration of them does not seem necessary. It is otherwise with disease of the membranes which cover the nerve-centres. The roots of the sensitive nerves pass through the spinal membranes, and have for their sheaths the prolongations of such membranes: consequently inflammation of these membranes gives rise, almost of necessity, to an inflammation of the posterior or sensory nerve-roots, with the necessary production of pain and hyperæsthesia. These sensory symptoms are always accompanied by spasm of the muscles, on account of the coexistent inflammation of the motor roots. The groups of symptoms which are thus produced have been sufficiently detailed under the head of Spasmodic Affections (see page 168). It seems only necessary here to point out that hyperæsthesia of the neck and face may be symptomatic of inflammation of the basal brain-membranes, and lateral hyperæsthesias and hyperæsthesias in the limbs, of inflammation of the spinal membranes.

PARÆSTHESIAS.

Under the name of paræsthesias may be grouped the almost innumerable disagreeable sensations which accompany functional and organic nervous diseases, and which are usually referred to the surface of the body, or more rarely to mucous tracts. Such are formications, prickling, a feeling as of the flowing of water or of the crawling of ants or other insects over the surface, itching, flushes of heat, waves of cold, etc. These symptoms may be due to hysteria, and are very common in women at the climacteric period, even when no distinctly hysterical symptoms are present. In rare cases of insanity they are psychical,—*i.e.*, of the nature of a delusion, the subject simply imagining their existence. They may be produced by various poisons. They are often the result of disease of the spinal cord, either simple congestion, myelitis, or

the curious condition which occasionally follows spinal concussion. When localized in one arm or in one side of the body, they may be produced by local brain-alterations, and occasionally they exist as prodromes of hemiplegia.

When paræsthesias are not hysterical or due to disease of the spinal cord, they are usually the outcome of some poison in the blood,—notably either the gout-poison or lead. In a case which had been under the care of most of the leading neurologists in the United States without plumbism having been suspected, the chief symptoms were an apparent insomnia, with horrible itching over the whole surface, and itching, with burning pains, in the urethra. A chemical examination of the urine and of the water habitually drunk by the patient demonstrated the existence of lead in each. The insomnia was undoubtedly due to the paræsthesia.

PAIN.

Pain is a symptom so variable and so universal that it is difficult to discuss it properly in connection with one class of diseases. Its importance, however, necessitates its consideration. In estimating the intensity and the diagnostic value of pain, it must be remembered that what may be called the pain-reaction varies almost indefinitely in individuals. A disease which in one person produces a veritable agony will in another cause but little suffering. It is stated that an old hardened cart-horse will quietly eat his oats whilst the operator is cutting down upon the thoracic duct, although a thoroughbred squeals, plunges, and becomes entirely unmanageable at the first touch of the scalpel. In man the differences in sensitiveness are as great as in the horse. Moreover, the statements of patients vary enormously in regard to pains of equal intensity. The hysterical person habitually overstates his sufferings: indeed, the peculiar exaggeration leads the experienced practitioner almost at once to a correct diagnosis. A very important assistance in the recognition of an hysterical or other exaggerated pain is to be obtained by noticing the disagreement between the words of the patient and the expression of the face. A well-timed compliment to a woman groaning in agony will often bring the reward of a momentary pleasant smile or expression of gratification and the consequent discovery of the meaning

of the alleged pain. Sometimes the words and the expression of the face perpetually give the lie to each other. Again, by skilfully distracting the attention of the patient, the pain or alleged tenderness can often be caused to disappear: thus, engaging the patient in active conversation may cause her not to notice firm pressure upon the spot which is alleged to be intensely sore.

As most local inflammations, ulcerations, or other organic changes produce more or less pain, it is essential that a careful examination be made to discover the existence of any local disease before the consideration of the nervous origin of the pain is entered upon. In the discussion which follows in this chapter it will be taken for granted that such examination has been made: *all the diagnostic procedures and rules given are founded upon the supposition that the existence of local disease has been excluded.*

The diagnostic import of pain in nervous diseases is best studied by dividing the body into three regions, and discussing each of these separately. These regions are the extremities, the trunk, and the head.

Neuralgic Temperament.—Before taking up the systematic consideration of the various localized nerve-pains, it seems necessary to say a few words in regard to the obscure but intolerable pains which may be included under the term *Neuralgia.* Formerly almost all forms of nerve-pains were spoken of as neuralgic, so that whenever there was a lack of obvious local lesion, such as of abscess, etc., the patient was said to be suffering from neuralgia. Most of the varieties of localized pains which will be treated of in subsequent sections have been split off from neuralgia, but there yet remain pains for which we cannot account, and to which, therefore, the term neuralgia is still applied. In many cases autopsies will show that the supposed neuralgic pain was caused by a definite lesion: thus, a clot forming near the trigeminal nerve-centre, or a spicule of bone hidden from external view pressing upon a nerve in such a way as to irritate it occasionally but not to provoke a defined neuritis, would give rise to pain that during life might have to be termed neuralgic. There is, however, a final group of cases in which neither during life nor after death are we able to detect any cause for the pain. It would seem that there may be a molecular change either in the

sensory nerve-centres or in the nerves themselves so fine as to escape our instruments, which predisposes the individual to suffer, so that a change of weather or other untoward influence too slight to be felt by the normal man causes a pain-storm. In a large proportion of cases neuralgia is undoubtedly of gouty or lithæmic origin; but it has seemed to me that we must acknowledge that there is also a general condition which may be known as the neuralgic temperament or diathesis. This is often inherited, but may be developed by prolonged bodily exhaustion or other causes. When once acquired, it may persist although the original cause has been removed. The pains which come to some persons in malarial anæmia probably are often neuralgic, but when the anæmia has been relieved, if the nervous system has been sufficiently long impressed, the pain-tendency becomes stamped upon it, precisely as the epileptic tendency becomes constitutional in a case of reflex epilepsy and persists after the removal of the original irritation. These neuralgic pains are to be recognized by their persistency, by the absence of cause, and by the excluding of all other sources of pain. I believe that the acquired or inherited neuralgic temperament frequently is closely connected with or complicates some of the pains hereafter to be described. Thus, I have seen cases of migraine in which, besides the definite attacks, there were not rarely seizures in various parts of the body, and still more frequently a complete departure of the migraine from its typical characteristics. Under these circumstances possibly a neuralgic temperament is superadded to an inherited migraine. The neuralgic temperament is undoubtedly closely allied to inherited gout; but the facts that the neuralgic temperament so often corresponds with the general neurotic temperament, that it is more frequent in women than in men, and that it is so often prevalent in dry neurotic climates and in persons free from gouty symptoms, indicate that the neuralgic temperament is something more than a gouty diathesis.

Reflex Pains.—Although in the following pages much will be said in regard to the occurrence of pains at a distance from the point of irritation, some general remarks upon the subject seem necessary. Precisely as there may be reflex disturbances of motility, so may we have reflex sensory disturbances. Usually the pain is felt in some region near to, or at least in relation with, the

seat of irritation. Thus, Anstie describes several cases of neuralgia in the urethra and testicles as the result of self-abuse. A facial pain caused by a diseased tooth may be due to a propagated neuritis; but that such pain may be reflex is shown by its not rarely occurring upon the side of the head opposite to the affected tooth, and also by the cases reported by Dr. Ferrier (*Thèse*, 1884, p. 376), in which facial neuralgia was produced simply by the irritation of tartar upon the teeth. Other not uncommon instances of neuralgic pains occurring near the seat of the irritation are the intestinal neuralgia which often accompanies impacted fæces, and the pain in the bladder which sometimes results from fissure of the anus. Although usually thus close at hand, the reflex neuralgic pain may be at a long distance from the irritated point and have no apparent connection with it. Thus, I have seen a mastoid pain, so violent as to lead to the diagnosis of disease of the bone and to a deep incision, caused by tapeworm in the large intestine; and in the thesis of Ferrier, quoted above, there are recorded instances of cervico-brachial neuralgia resulting from a diseased tooth. The recognition of the character of the pain in these cases depends upon the acuteness of the physician in perceiving the absence of other causes and the presence of the irritation.

PAINS IN THE EXTREMITIES.

A persistent pain occurring in the legs or in the arms, and not dependent upon obvious local disease, may be due—

1. To gout or rheumatism.
2. To neuritis.
3. To chronic metallic poisoning.
4. To disease of the spinal cord.
5. To neuralgic or malarial cachexia or other obvious or obscure cause.

In deciding to which of these categories any individual case belongs, the diagnosis must, to some extent, be reached by the process of exclusion, and aid must be obtained from the concomitant symptoms. The character of the pain, although of some importance, is not decisive, for the same quality and severity of pain may arise from various causes, and in different individuals the same cause varies almost indefinitely in its pain-effects.

Pain is mobile, shifting, or darting, or it is fixed in one spot.

Fixed Pains.

A permanent fixed pain may be the result of a neuritis, but is in the great majority of cases the expression of gout or of rheumatism. This is especially the case when it is associated with tenderness upon pressure or upon motion. A fixed pain may be the result of a sprain or other injury; but a traumatism may cause the fixation of a general rheumatic irritation in the injured part, so that care is sometimes necessary to prevent being deceived in the diagnosis. If a rheumatic seizure be very sudden, and occur at the time of making an exertion, the pain may be supposed to be the result of an injury. Thus, I saw, not long since, a grocer who had been under professional care for nearly a year for strain of the back. I found, however, that at the time of the sudden coming-on of the pain he was lifting only a few pounds, whereas he had been accustomed for years to lift occasionally a barrel of flour without injury. There was also a distinct history of exposure to damp, with increase of the pain at night and on change of the weather. On putting the patient on anti-rheumatic treatment, relief was soon afforded. In another case a gentleman springing from a wagon was perfectly well when he left his carriage, but on reaching the ground had a disabling pain at the insertion of the tendon of the right patella, and supposed that he had wrenched his knee. It turned out, however, that the attack was rheumatic.

The mere permanence of the gouty or rheumatic pain sometimes misleads the practitioner, especially when it affects the extremities symmetrically. A rheumatic pain may continue in a single spot for months, and even for years, and may be located in exactly corresponding portions of opposite limbs. A rheumatic pain is to be distinguished from a pain of nerve-origin by the tenderness on pressure and on active movement, and also by the pain which is elicited when passive movements are so made as forcibly to stretch the affected muscle.

Mobile Pains.

The diagnosis of the nature of mobile pains is often one of much difficulty. In deciding the nature of such a pain the first consideration is as to whether it is unilateral or bilateral. The lesions

of the spinal cord itself which produce pains are almost invariably bilateral. In the beginning of a syphilitic or other localized disease of the spinal membranes, one side may be affected, and consequently a unilateral pain be produced by involvements of the spinal roots of that side. It is rare for a localized meningeal affection to be situated so low as to implicate the nerves going to the legs: consequently, only in the rarest cases is a unilateral pain in the leg due to disease of the cord or of its membranes. In like manner, a pain situated in one arm and not in the other is practically never due to disease of the cord itself, and only in rare instances to an affection of the membrane. A unilateral pain in an extremity is therefore, except in the rarest of cases, not due to organic disease of the nerve-centres or of their membranes.

Pains which are the result of a general toxæmia are apt to be bilateral, because the nervous system on each side of the body is equally under the influence of the poison, but, as already stated, the constitutional disorder may for long periods of time expend itself upon one point. If the toxæmic unilateral pain be darting, shooting, or shifting, it is due to the constitutional affection influencing a nerve-trunk, so that it may be laid down as a general diagnostic rule, whose exceptions are exceedingly infrequent, that *a unilateral pain either in the arm or in the leg is due to an inflammation of the nerves themselves*, which neuritis is generally the result of a constitutional disorder,—alcoholism, gout, rheumatism, syphilis, etc. In the examination of a case suffering from a unilateral pain in the leg, pressure upon the sciatic nerve at its emergence from the pelvis, upon the popliteal nerve, or upon some of the smaller nerve-trunks, will, as a rule, detect the existence of distinct tenderness, or such tenderness of the nerve-trunks will be made manifest on stretching the nerve by forced movements of the limbs. In the arm the brachial plexus or its branches will be found sore. Under these circumstances the diagnosis of neuritis becomes plain, and in the majority of cases such neuritis is rheumatic or gouty.

Theoretically, we should expect to find cases of unilateral nerve-pains in the extremities without tenderness of the nerve-trunks and without obvious cause,—pains parallel to those so frequent in the head,—but, for some reason as yet unknown, these obscure nerve-storms are very infrequent in the extremities.

One of the most characteristic symptoms of posterior sclerosis of the spinal cord is the pain. As this pain may precede by many years other manifestations of the disease, its study is of great importance from a diagnostic point of view. In a majority of cases the legs are the first portions of the body to suffer. In the rare instances in which the selerosis commences in the upper portions of the cord the arms are primarily implicated, and even the region of the face may be the first to be invaded. The pain is usually very severe and momentary. It is variously described by the patients as shooting, darting, as a feeling as though lightning were shooting through the part, or as though a red-hot wire or a sharp dagger were thrust through the limb; rarely it is burning. In some cases these so-called fulgurant pains occur continually; in other instances they come on in paroxysms, which may in their severity amount to a veritable crisis. Even if they have been habitually present for a length of time, they may cease for a time. Sometimes they distinctly follow the distribution of the nerves. They are commonly felt severely in the neighborhood of joints: thus, the inside or the outside of the knee, or the inside of the ankle, is often the seat of violent darts. Usually they are not associated with redness or any soreness, and often the patient seizes the affected part forcibly and obtains by the pressure some relief. In some instances, however, a certain amount of redness and tenderness is present during the paroxysms of the pain, and in exceedingly rare cases trophic eruptions occur. The peculiarity of the pains of locomotor ataxia is sufficient to enable us to recognize their character, even when other symptoms of the affection are not perceptible, although the diagnosis cannot be considered as positively established until the loss of the knee-jerk, or some other symptom of the disorder, is developed. The pains are always bilateral, are not increased by motion, and are not accompanied by a persistent soreness of the part. If there be any tenderness, it is only during or immediately after the pains. Wandering gouty pains sometimes simulate the pains of locomotor ataxia, but are never so severe or so persistent, unless there be a gouty neuritis, which will be at once revealed by the persistent tenderness. Moreover, the shooting gouty pain is always associated with other marked evidences of the diathesis.

Although bilateral, a moving pain, accompanied with tender-

ness of the nerve-trunks, with pain on motion, passive or active, is never the direct outcome of a disease of the spinal cord. It may be a symptom of a descending neuritis due to an involvement of the nerve-roots in diseases of the spinal membranes. Moreover, there is much evidence to show that descending neuritis occurs in certain diseases of the spinal cord itself. Under such circumstances tenderness of the nerve-trunks is naturally to be expected; but it must be remembered that such tenderness does not develop until late in the disorder, and then only in rare cases.

In various diseases of the nerve-centres, contractures, especially in lateral sclerosis, may exist, and the attempt at forcible extension of the limb may give rise to pain. This pain, however, must not be looked upon as the result of hypersensibility of the affected muscles, but as similar to that which would be caused by attempting to stretch beyond its normal length a muscle which has not undergone contractures.

TRUNKAL PAINS.

Reflex Pains.—Many diseases of the muscles, membranes, or viscera of the trunk are accompanied by pain. A discussion of such pains lies without the province of the present work, but it may not be improper to say a few words in regard to certain of them which are reflex in origin, and which, although due to local disease of viscera, are situated at such a distance from the point of organic alteration that they are liable to be mistaken for pure nervous or neuralgic pains. The most important of these reflex pains are those produced in the shoulders or in the back by diseases of the liver or of the stomach. A peculiar fixed pain in the upper portion of the right shoulder is a not very uncommon symptom in congestions and other diseases of the liver. The pain of gastric ulceration is usually referred to the back, between the scapulæ. I have known it to be in the lumbar region, and occasionally it is high up in the shoulders. I have seen the abdominal pain which is produced by the escape of a meal into the abdominal cavity through a sudden perforation of the gastric wall entirely masked by a horrible mortal agony, referred by the patient to the top of the left shoulder, and, what seems most extraordinary, markedly increased by movements of the body. At the height of the agony any alteration of posture produced a violent, excru-

ciating spasm of the scapulary and dorso-cervical muscles. In this case the ulcer was not suspected during life.

Rheumatic Pains.—It seems hardly necessary to say much concerning rheumatic pains in the muscles of the trunk, but it is perhaps proper to call attention to the fact that sometimes they are excessively severe, and appear with such abruptness that they may be supposed to be the result of traumatism. They are always accompanied by excessive pain on motion, and can scarcely be mistaken for other idiopathic affections.

Pains of Chronic Fibrous Inflammation.—A form of back-pain which is not infrequent as the result of injury is a persistent aching, with a marked and pronounced sense of weakness of the part, and sometimes stiffness and pain on movement. This condition, which may last for years, is, I believe, not a nerve-pain, but is due to a very slow chronic inflammation of the fibrous tissues which bind together the vertebræ. It is especially apt to be present in the lower portion of the back.

Toxæmic Pains.—Toxæmic pains, due either to gout or to malaria, are liable to attack any portion of the body. They may locate themselves in the neighborhood of the kidneys, and under these circumstances closely simulate the pains of renal colic. They can usually be distinguished by their not being so severe or so persistent, and by their not shooting into the genital organs. Not much aid in the diagnosis can be obtained from an examination of the urine, since crystals are often not present in the urine in cases of renal calculi, and the elimination of uric acid often accompanies the gouty pain-paroxysm. In a malarial case, a more or less regular periodicity will usually betray the nature of the disease.

Herpetic Pain.—A violent stinging pain shooting along the course of the intercostal nerves, or located in irregular spots in the front of the body, not rarely precedes, but perhaps more generally accompanies, the eruption of herpes zoster. Both the eruption and the pain are due to inflammation of the intercostal nerves, in most cases of a gouty nature.

Pain of Vertebral Caries.—A fixed, unaccountable pain in the shoulders, or very rarely in the lumbar region, may be the first obvious symptom of a commencing spinal caries. I have so frequently seen this pain of sympathetic inflammation of the nerve-roots precede the more manifest symptoms of spinal caries that

I look upon it as a forerunner of very serious disease. If an individual be of such age and physical character as to bring an attack either of syphilitic or of tubercular disease of the vertebræ within the range of probability, a fixed, apparently causeless, severe neuralgic pain either in the shoulder or in the front lumbar region should be viewed with the greatest suspicion, and should lead to the most careful study of the case.

Girdle Pain.—The girdle sensation, so called, is a feeling as though a band were tightly drawn around the body. If really present, and not imagined by the patient after the suggestion by the physician, it, I believe, always indicates organic disease of the spinal cord or of the nerve-roots. When not amounting to absolute pain, it is usually due to chronic myelitis or to spinal sclerosis. When very severe, it may be caused by inflammation of the nerve-roots, due to cancerous, syphilitic, or tubercular degenerations of the spinal membrane. In some cases this girdle pain instead of being around the body encircles some portions of the legs. Under these circumstances it is bilateral and symmetrical. I cannot remember to have seen it lower than the garter line.

Pain-Crises.—The most important, as well as the most severe, of all the nerve-pains connected with the human trunk are the so-called pain-crises which occur in locomotor ataxia. When once established, these crises usually persist through the whole course of the affection, but they are frequently among the earliest of prodromes, and may by many years precede the more common symptoms of the disease.

In a remarkable case now under my care in the University Hospital, in which the symptoms followed an injury to the foot, and are apparently the result of an ascending neuritis, moderately severe gastric pain-crises occur simulating those of locomotor ataxia. The symptoms in the case are and have been violent pain, gradually extending up the leg, and thence to the half of the body of the same side, at present very severe in the arm and face; marked tenderness over all the nerve-trunks which are the seat of pain; exaggeration of the patella-reflex upon both sides, but more markedly upon the side most affected; contraction of the field of vision, with atrophy of the nerve much more pronounced in the eye upon the affected side; failure of strength of

the affected leg, with some contractions of the muscles and pain on stretching the foot; distinct atrophy of the muscles of the affected leg, with modal changes in their electric contractility; upon the diseased side great coldness of the foot and lower leg, with a somewhat similar condition in the forearm.

A pain-crisis consists in its essential parts of a paroxysm of pain as violent as human nature can endure, accompanied by excessive functional disorder of the part attacked, developing usually with great rapidity, but disappearing as rapidly as it appeared, and associated with a condition of undisturbed functional activity of the affected viscus between the paroxysms. The crisis may at first return only at long intervals, but as the disease progresses it comes on more frequently, and often more severely, until at last in some paroxysms syncope alone brings relief. During the paroxysm there is no fever; but the patient is often left in a condition of profound exhaustion.

The most important of the pain-crises are the muscular, the gastric, the visceral, the rectal, the urinary, the genital, the cardiac, and the laryngeal.

The *muscular* pain-crises are extremely rare, but have been described in two cases by Dr. Pitrès (*Prog. Méd.*, July 12, 1884). They consist of a feeling of lassitude, deepening into an excessive muscular weariness and soreness like that which follows violent exercise in one unaccustomed to it, and at last entirely paralyzing for the time being the affected muscles. They appear to attack more usually the lumbar group, and to make it impossible for the patient to assume an erect position. They are said to last from a few hours to many days.

The *gastric* crises appear to be the most frequent of any of the pain-paroxysms of locomotor ataxia. They are characterized by violent pains having their focus in the epigastric region and radiating in all directions, laterally, upward, and downward, until at times they seem to fill with agony the whole abdomen and chest. The pains are sometimes spoken of as constrictive, more usually shooting like a dagger-thrust; not rarely they are burning. They are generally increased by the ingestion of food, and are always accompanied by nausea and excessive vomiting and the rejection of everything that is put into the stomach. The vomiting is repeated many times an hour, and is accom-

panied by horribly distressing retching. After the stomach has been emptied, glairy or ropy mucus is ejected: this soon becomes greenish from the presence of bile; and in severe cases streaks of blood appear in the mucus: more rarely there is abundant coffee-ground vomiting or even pronounced hæmatemesis. (See case, Vulpian, *Maladies du Système nerveux*, vol. i. p. 267.) In some cases the focus of the pain is in the neighborhood of the umbilicus, when the crisis might properly be spoken of as *intestinal*. Occasionally large quantities of gas form in the gastro-intestinal tract, and produce a very obstinate meteorism, which is itself more or less painful, and is scarcely diminished by the incessant discharge of gas by the mouth or rectum. This paroxysm of atrocious suffering may last from one to a few hours, or even for days, interrupted, it may be, by syncope, and finally leaving the patient in a condition of profound collapse. It is sometimes accompanied by a hyperæsthesia of the epigastric region so excessive that the mere contact of the hand will provoke violent pains. During the attack it is absolutely impossible for the patient to take food, but usually the paroxysm ends abruptly, and food is immediately desired and is digested without difficulty. When the force of the paroxysm expends itself upon the stomach there is no disturbance of the bowels, but if enteralgia be severe there is usually with it an abundant discharge of bilious or mucous or serous stools. In such cases the patient may assume the facies of cholera, the likeness being made complete by the loss of the voice, the suppression of urine, the extreme coldness and cyanosis of the body, and even the presence of cramps. Vulpian reports a case of death during such a paroxysm.

The gastric or gastro-intestinal crisis is distinguished from similar attacks due to disease of the stomach or the bowels, by the suddenness of its development; by the excessive severity of the symptoms; by the absence of the ordinary signs of organic disease of the stomach or intestines; by the abruptness of the termination of the paroxysm; and by the complete performance of the normal functions between the attacks. It might be simulated by hysterical gastralgia with vomiting, but in the latter disease the symptoms are not so severe as in the crisis, and are attended by globus hystericus, great emotional disturbance, convulsive movements, anæsthesias, or other well-recognized symp-

toms of hysteria. The occurrence of hæmatemesis in gastric crisis must always suggest the presence of gastric ulcer, but between the paroxysms the symptoms of such ulcer ought to be apparent if the lesion really exist.

Much more rarely than the stomach are other abdominal viscera the seat of pain-crises in locomotor ataxia. In some cases true *rectal* crises occur, with violent pains of a cutting, shooting, burning character radiating from the rectum in every direction. Not rarely the patient complains bitterly of a sensation as though the rectum were filled up by an enormous body heated to redness, burning and scorching every part near it. (See Trousseau.) Among the most painful of these abdominal crises are those connected with the *urinary* organs. The symptoms may resemble so closely those of renal colic as to make the immediate diagnosis almost impossible. Thus (M. Raynaud, *Arch. Gén. de Méd.*, October, 1876), a man attacked in the streets of Paris was brought to the hospital bent double, suffering from a furious pain in the belly radiating into the lumbar region along the tract of the ureters, and accompanied by retraction of the testicle. An incorrect diagnosis of renal colic was at first made. More frequently the pains are in the urethra, where they manifest themselves as intense burning, or as lancinating darts of agony shooting through the whole length of the urethra and simulating the pain of calculus. Usually at the same time there is great distress in the bladder, and an incessant desire to pass urine, with the emission of only small quantities with great straining and without relief. These pains in some cases occur in paroxysms similar to those of the gastric crisis; in others they are more persistent, so that the patient suffers from almost constant distress. Under these circumstances there may be anæsthesia of the ureter and of the bladder, with consequent retention of urine and ammoniacal fermentation. In some of these cases the urine becomes loaded with phosphate, and the mistaken diagnosis of phosphatic calculus might be readily made.

Genital Crises.—Genital disturbance is almost universally present in locomotor ataxia. Usually it takes the form of loss of functional power. It is in my experience very infrequent for this depression to be preceded by sexual excitement, but Trousseau relates the case of a man in whom the first symptoms of the disorder

were excessive lust, and a strange power of repeating coitus a great number of times in rapid succession. Associated with such sexual excitement is usually excessive quickness of emission during the act,—a quickness which augments until it amounts to a veritable spermatorrhœa, the semen being discharged upon the slightest provocation, and a true impotence resulting.

In rare cases veritable genital crises occur. These are of two characters. In one, violent paroxysms of pain centre in the testicles and shoot along the penis to its head, or, in the female, burn and bore in the ovaries, the labia minora, and the clitoris. In these paroxysms the agony is only a little less than that of the gastric crisis. In other cases spontaneous venereal orgasms replace the pain-paroxysm, either in the male or the female. Thus, in a case reported by Prof. A. Pitrès, a woman suffered with frequent paroxysms, commencing with a sense of vibration in the vagina, followed at once by erection of the clitoris, voluptuous sensation, and rapid orgasm. The venereal crises often occurred four or five times in the twenty-four hours. After the lapse of four years they became associated with fulgurant pains. Four years later a gastric crisis occurred, and the other symptoms of locomotor ataxia slowly developed. The close connection between the venereal and pain paroxysms was shown by the fact that in the later years a violent attack of fulgurant or gastric pains was always ushered in by an erotic crisis. In another case reported by Pitrès the venereal paroxysms preceeded the fulgurant pains by ten years.

Laryngeal Crises.—The laryngeal crisis is a very rare phenomenon in locomotor ataxia. Briefly spoken of in 1862 by M. Bourdon, it has been fully described by M. Féréol (*Gaz. Hebdom.*, February 12, 1869) and by M. Jean (*ibid.*, July 7, 1876). It consists of violent paroxysms of coughing, with great laryngeal disturbances of respiration, atrocious fulgurant pains in the shoulders and along the spinal column, and pronounced symptoms of asphyxia. The face is red, intensely congested, and finally cyanosed. The cough, furious, hoarse, grating, is rapidly repeated in paroxysms, ending in a raucous inspiration like that of whooping-cough. The expectoration is of a scanty saliva-like secretion, or rarely of little pellets of mucus stained with blood. The partial expirations, abrupt and jerky, follow one another with convulsive

haste, to be succeeded by a prolonged blowing inspiration. When the paroxysm is severe the dyspnœa is extreme, and the urine and fæces may be passed involuntarily. The paroxysms occur spontaneously by day or by night, but are also produced by draughts of air, or by the swallowing of hot drinks or food, etc. There is no asthmatic dyspnœa between the paroxysms, although a permanent emphysema may be produced by the strain of the violent efforts at breathing. The attack usually begins and ends abruptly. In the case reported by M. Jean, spasm of the muscles of the pharynx finally prevented swallowing altogether; asphyxia alternated with syncope, until true coma appeared, to end in death. At the autopsy posterior spinal sclerosis was found; but, as there was also a pronounced lesion in the medulla, it remains doubtful how far the laryngeal symptoms were the result of the spinal degeneration.

Cardiac Crises.—In 1879, Vulpian called attention to the frequency of valvular disease of the heart in locomotor ataxia, and his observations have since been confirmed by both German and French writers. Insufficiency of the aortic valve appears to be the most frequent lesion; but Grasset shows by a report upon twenty-four cases that the heart-lesions are various. This is confirmed by A. Jaubert (*Thèse*, Paris, No. 137, 1881), who further makes it plain that not only the valves but also the heart-walls may be affected.

It is at present uncertain whether the cardiac lesions are due directly to the disease of the nerve-centres, or whether both the nervous and the cardiac affections are the result of a common cause. That the changes in the heart are not trophic or parallel to those which occur in the joints of the ataxic is strongly indicated by the fact that in a number of Grasset's cases the cervical spinal cord was not implicated. The close connection between syphilis and locomotor ataxia on the one hand, and between syphilis and arterial degenerations on the other, suggests that the two diseases are frequently the result of a common cause. In many cases the cardiac affection comes on very insidiously, and symptoms may not appear until long after serious lesions have been developed: therefore the practitioner should habitually auscult the heart in cases of posterior spinal sclerosis. In some cases violent cardiac crises occur. It is uncertain whether they are always associated with lesion either of the heart-valves or of the heart-walls.

They have been present when there were no sufficient physical signs to justify the diagnosis of cardiac lesion, and it is therefore probable that a cardiac crisis may represent a nerve-storm similar to that of a gastric crisis. Charcot has noticed that there is often, if not always, a permanent acceleration of the pulse in these cases. The symptoms of the cardiac crisis are similar to those of angina pectoris,—namely, violent pain in the region of the heart, associated with great dyspnœa, intense distress, and irregularity of the pulse, with or without intermission of the heart-beats.

HEAD-PAINS.

The causes of headache are almost innumerable, and to discuss them fully would require a volume. I shall therefore confine my attention to a consideration of the chief varieties of headache not connected with acute disease, such as fevers, pneumonias, etc. In treating any individual case of chronic headache, the first vital decision is as to the cause of the head-pain. For this reason it seems proper in this work to view headache chiefly from an etiological stand-point. The character of headache varies excessively. It is sometimes deep-seated; sometimes superficial; sometimes a distress; sometimes a violent pain; sometimes a heavy ache; sometimes an acute throbbing; now it fills the whole cranium, again it radiates over the surface, or settles in some one point. It is paroxysmal or constant, shifting or fixed. Unfortunately, the character of the headache varies in different individuals with the same cause: thus, even the headache which is the result of an organic brain-lesion is different in different patients. It is impossible to make the diagnosis as to the nature of the headache from a study of the headache itself: only by a consideration of the concomitant symptoms and in many cases by the process of exclusion are we able to arrive at an approximately correct view. Cases not a few offer themselves in which the nature of the headache is finally made out only by studying its response to therapeutic agents. Nevertheless, something can often be inferred from the seat of the pain and from its character, and therefore I shall point out, as far as may be, peculiarities in individual headaches. The best arrangement of headaches for clinical study that I have been able to formulate is comprised in the following scheme:

1. Organic headaches, or those which are due to disease of the brain-membranes or of the brain-substance.

2. Toxæmic headaches, or those which are due to the circulation in the blood of some poison, either engendered within the body or received from without.

3. Sympathetic headaches, or those which are due to some peripheral lesion, which acts apparently through the nervous system without the formation of a poison.

4. Headaches which are not included in the other groups, and to which the name of nervous may be given, with the understanding that the title carries no etiological significance. In this group are placed many headaches of whose ultimate cause we are ignorant.

Although I have separated these four groups of headaches, it must be distinctly understood that no group is characterized by a constant peculiarity in the head-pain, and that in the diagnosing of an individual case it is often necessary to pass in review all the sub-groups, of which I shall speak later.

It is a matter of the gravest importance that the pain of an acute *glaucoma* shall not be mistaken for headache. The pain, which may develop with great suddenness, often centres in the eyeball, but may seem to have its chief focus in the supra-orbital notch: not rarely it shoots over the forehead and into the cheek and temple, reaching even to the occiput, and filling the whole side of the head with agony. If, as usually happens, there is fever, with severe vomiting, the patient may be thought to be suffering from a bilious or malarial attack, and the eye be irretrievably damaged before the true nature of the paroxysm is discerned. This can be avoided by paying attention to the following points: the eye shows evidences of inflammation in congestion and swelling of the conjunctiva and even of the lids; the cornea is somewhat misty, presenting the appearance sometimes spoken of as "steaminess," and its sensitiveness to the touch of a camel's-hair pencil is diminished; the pupil is sluggish, often somewhat dilated; on palpating the two eyeballs simultaneously with the forefingers, the affected eye is felt to be the harder, and the patient often complains of a sense of tension in the ball; vision is less acute in the affected than in the sound eye. In case of doubt it is the duty of the practitioner to call in an oculist at once.

Toxæmic Headache.

The most important varieties of toxæmic headache are malarial, rheumatic, gouty, uræmic, diabetic, alcoholic, and caffeinic.

Malarial Headache.—A headache may occur in a malarial subject as a secondary result, produced by the anæmia or by the disorder of the gastro-intestinal tract, etc. Such headaches are anæmic, gastric, etc., rather than truly malarial. The specific malarial headache occurs in paroxysms at more or less regular intervals. It almost invariably takes the form of the so-called "brow ague," in which an intense pain rapidly develops at fixed hours in the immediate neighborhood of one supra-orbital foramen. This pain lasts from five to ten hours, is often of frightful intensity, and may or may not be associated with fever and sweat or other indications of a malarial paroxysm. It is a malarial paroxysm which is to be recognized by its form, and especially by the regularity of its recurrence and by its rapidly yielding to quinine when given in sufficient doses. It must be remembered that it is often necessary to administer as much as thirty grains of quinine just previous to the expected paroxysm in order to obtain distinct relief.

Rheumatic Headache.—Rheumatic headache is not infrequent. It usually takes the form of heavy aching pain, worse at night and on the approach of storms, and accompanied by more or less soreness of the scalp: under these circumstances the rheumatic irritation undoubtedly expends itself upon the fibrous tissue of the scalp. In other cases the pain is severe, sharp, and shooting, passing into the jaws or coursing over the forehead: such pain is the expression of a rheumatic neuritis affecting the branches of the trigeminal nerve. The rheumatic headache may be without any characters indicating its nature. In a sculptor who was about to abandon his profession on account of excessive intractable headaches, I found that the headaches had occurred only during the time when the artist was working upon the modelling in wet clay of a very large composite life-size group, and, not being able to make out any other explanation of the headaches, I put the patient on anti-rheumatic treatment, with the most satisfactory results.

Lithæmic Headache.—Lithæmic or gouty headache in its

usual form is dull and heavy, and often worse on rising in the mornings. It may, however, be acute, and I have seen it excessively violent: in one case, for a series of years there were headaches whose cause could not be made out, and whose violence was so great as to make life unendurable. Not a day passed without them, and much of the time the head-pain was an agony. In this case the headaches finally became associated with attacks of loss of consciousness, which closely resembled petit mal, so that I was led to the diagnosis of an organic lesion of the brain or its membranes. Finally, all the small joints of the body and many of the large were simultaneously attacked with a furious sudden general gout, with enormous deposits and permanent disablement. The headaches were greatly relieved by this outbreak, but have reappeared from time to time, although the joint-lesions have progressed so that the patient is entirely crippled. My own belief is that there was originally a gouty thickening of the dura mater with deposit, so that the headaches were not simply the result of gouty irritation, but were due to a gouty organic lesion.

Uræmic Headache.—The uræmic headache may take almost any form, and the diagnosis must be made out by detecting the kidney-disease. Some years since, a patient was brought to me who was suffering from a unilateral frontal headache, which always commenced from one to two hours after rising, and continued to grow more intense until the man went to bed, when it disappeared. Examination of the urine revealed the nature of the trouble. In the pre-albuminuric stage of gouty kidney, when the heart is somewhat hypertrophied, the vessels more or less rigid, and the arterial tension raised, headache is a common symptom. How far under these circumstances the headache is due to retention of matters in the blood which ought to be excreted, how far it is the result of the increased arterial tension, how far it is gouty in its nature, often cannot be made out. Dr. S. Weir Mitchell speaks somewhere of having seen cases in which repeated headaches preceded hemiplegia. It seems probable that these headaches were either gouty or uræmic, and were only by accident associated with the subsequent rupture of a blood-vessel.

Alcoholic Headache.—Headache is a common symptom in chronic alcoholism; in some cases it may be due to the direct irri-

tation of the brain-membranes by the alcohol; but usually it appears to be secondary to the gastro-intestinal irritation.

Caffeinic Headache.—A very common headache is that which I have called caffeinic, because it is the result of the excessive use of coffee or tea. The subjects of these headaches are almost always of neurotic temperament; not rarely they suffer from migraine or some form of nervous headache. It must be borne in mind that even a small amount of coffee may, in such persons, produce disabling head-pains. Overworked seamstresses and sewing-women often supply a lack of food and strength by an excessive use of tea. Under these circumstances severe cephalalgia and other nervous symptoms are certain to occur. There is no method of determining in any individual case that the headache is due to the use of tea and coffee except by noticing the effect of suspending these beverages. Before a decisive result can be considered to have been reached, total abstinence must have been enforced for at least three weeks, since when the train of morbid symptoms has once been set in motion very small amounts of the beverage suffice to keep it moving.

Gastric Headache.—Headache from disorder of the liver is frequent. It can hardly be separated from that which is produced by gastric derangement, although in some cases the gastric headache-pain is evidently reflex, due to irritation of the peripheral nerve-filaments in the stomach by exceedingly acid and acrid contents. This acid-stomach headache is usually frontal, and is often accompanied by sudden blindness and dizziness and acid eructations. Its true nature is revealed by the immediate relief which is afforded by the use of large doses of aromatic spirits of hartshorn. On the other hand, a dull, heavy headache which often accompanies indigestion and hepatic torpor is probably the result of the absorption or retention of poisonous organic products. It is usually frontal, but occasionally is referred to the region behind the ears or to the occiput. It may be associated with defective vision, giddiness, and great depression of spirits.

Diabetic Headache.—Headache is not rarely present in diabetes. When it occurs in an advanced stage with great severity it is of special importance, because it is frequently prodromic of diabetic coma. Under these circumstances it is usually accompanied by dizziness, muscular pains, gastric distress, and dis-

ordered mental action, as shown by incoherent talk or by actual delirium.

Cardiac and Pulmonic Headaches.—Violent headache often accompanies diseases of the heart and lungs which are sufficiently severe to interfere either with oxidation of the blood or with the circulation. In cases of obscure chronic headache, especially in children, practitioners should always carefully examine the condition of the heart.

Sympathetic Headaches.

Pains in the head are in rare instances the result of comparatively distant irritations: thus, there are cases on record in which lancing the gums or removing a diseased tooth has relieved a severe and perhaps long-existing headache.

Headaches of Eye-Strain.—Severe headaches frequently result from eye-strain. The head-pain is produced by a disorder of accommodation, or by an insufficiency of one of the ocular muscles. Although very frequently the facts connected with the pain are suggestive of its cause, yet the headache of eye-strain has no fixed determinate character. It is usually frontal or in the region of the eye, but this position is not always selected. In a case reported by Dr. William Thomson (*Med. and Surg. Reporter*, 1874), the headache finally assumed characters exactly simulating those of the most typical migraine, the paroxysms commencing with an attack of partial blindness involving half the visual field, followed by severe pain in the head lasting many hours, accompanied by nausea and great general depression. In almost all cases the pain is greatly aggravated by the use of the eyes, and in the earlier periods of its history only follows such use; finally it may come on at all times, and often apparently spontaneously. It is apt to be very severe in the mornings after an evening spent at the theatre or other place of amusement where the lights are very bright. Sometimes the pain is not confined to the head, but radiates down the back. The difficulty of diagnosis is often aggravated by the fact that the headache of eye-strain is especially common in neurotic subjects, and that it not rarely coexists with head-pain of other character. The conjunctival symptoms, although not constant, are characteristic: they consist of chronic irritation, with intense redness and velvety appearance

of the mucous membrane and of the tarsus. Although in many cases the symptoms are sufficiently definite to lead to a strong suspicion of the cause of the headaches, a positive decision can be reached only by a careful examination of the eye by the oculist, and the counteraction of any defects that may be found.

Nasal Headache.—Headaches may be the result of disease of the nasal mucous membrane. Prof. Harrison Allen concludes that there are three kinds of these nasal head-pains, which he denominates respectively the reflex, the neurotic, and the inflammatory. The reflex headache is almost entirely restricted to the forehead, the temple, and the vertex. By drawing the index finger across the face from the middle of the nose to the temple, and thence in some cases to the parietal eminence, the patient often indicates the seat of the pain. In severe attacks pain sometimes radiates to the vertex and even to the nape of the neck, and then often nauseates and simulates migraine. Sometimes the point of pain is narrowed to a minute focus or spot. A very characteristic symptom is the marked increase of the headache upon the slightest exacerbation of the catarrh. A diagnostic symptom is tenderness of the inner wall of the orbit when pressed upon by the finger; or a probe passed into the nose causes an immediate access of pain when it reaches the right middle turbinate bone. The disappearance of the catarrhal reflex headache when the nasal catarrh is cured is the strongest proof of its nature. The neurotic nasal headache of Prof. Harrison Allen comprises cases in which highly neurotic individuals complain of violent pain in the throat, the ears, the back of the head, or even the pharynx, or of various distresses about the head, as a result of a moderate degree of local nasal or pharyngeal disease. Dr. Allen further says that he has never seen catarrhal headaches of inflammatory origin except in acute congestion or inflammations of the frontal sinuses: the pain is of high grade, is as a rule confined to one side, and subsides after the local application of leeches.

Nervous Headache.

Under the title of nervous headache I shall group the so-called anæmic headache, congestive headache, the headache of brain-exhaustion, the hysterical headache, migraine, and certain

rare headaches whose nature is completely obscure, but which may be designated by the misnomer of idiopathic headache.

Anæmic Headache.—The headache which is seen in those who are suffering from well-marked anæmia following malaria, bleeding piles, etc., has in itself nothing that is peculiar or characteristic. It is often brought on or aggravated by use of the brain, and in this way is related to the headache of brain-exhaustion. Very frequently during the attack the face will flush and the eyes redden, and the patient complains of a sense of fulness in the head. In these symptoms the headache resembles the congestive headache. As in many other of these forms of headache, the diagnosis is to be made out by discovering the existence of the disease or condition which produces the headache, and by the relief which follows the cure of the parent affection. The headache which is sometimes associated with fatty heart is probably due to an improper supply of blood to the brain, and may properly be considered to be a variety of anæmic headache. It must be borne in mind, however, that palpitation of the heart and cardialgic disturbance may be prominent symptoms in anæmia, and mislead the diagnostician into supposing that a cardiac lesion exists.

Congestive Headache.—Congestive headaches, or headaches from active hyperæmia, are, I think, extremely rare, unless after exposure to the sun or some other immediate exciting cause. They are to be distinguished by the severity of the throbbing pain, by the sense of pressure and weight in the head, by the suffusion in the face and eyes, and by the strong pulsation in the carotid. The pulse is usually full and strong, and the cephalic symptoms may go on to the appearance of hallucinations, and even to the production of coma or delirium.

Hysterical Headache.—In many cases of hysteria the patient suffers much from violent pains in the head, of varied character.

Almost characteristic of the temperament is the so-called *clavus*, a pain situated in the middle of the top of the head in a point so small that it can almost be covered with the point of the finger. The hysterical headache is apt to be increased at the menstrual period, and to be suddenly removed by pleasurable mental excitement.

Headache of Exhaustion.—In exhaustion of the general

nervous system, from such generally acting causes as severe conjoined mental and bodily exertion, nursing, depressing emotions, sexual excesses, etc., or in the limited exhaustion of the brain-centres from excessive intellectual work, the patient commonly suffers from a sense of weight at the top of the vertex, or from a heavy, dull, oppressive, deep-seated cephalic distress. This form of headache is often associated with insomnia, and is always increased by any intellectual effort.

Migraine.—Under the name of migraine, or megrim, are included very numerous cases, which, while they have much in common, vary greatly in the development of their symptoms. The essential feature of the affection is a paroxysmal headache, which in the great majority of cases appears first at early puberty and continues in women up to the menopause, or in men to advanced middle life. In its details the paroxysm varies in different individuals, but usually conforms more or less to the following type. For some hours before the attack the patient suffers from malaise, often with chilliness and a sense of languor, or in rare cases experiences a condition of peculiar emotional and mental activity. The attack may or may not be ushered in by distinct prodromes. The pain is unilateral in the great majority of cases, and is referred to the frontal region, having the focus at or about the supra-orbital foramen, or more rarely in the eye itself. It comes on gradually, becoming more and more intense for hours, until finally it is unbearable. It is generally described as boring in character, often throbbing, and only in very rare instances as shooting into the jaws and the neck. Sometimes the occipital region may be the seat of the pain. About the time that the pain reaches its greatest intensity, nausea followed by vomiting develops. The vomiting is usually repeated, and is attended with great bodily depression. The matters ejected are the contents of the stomach, followed by mucus and bile. Apparent relief often follows the vomiting. In some cases the patient now falls asleep, and wakes free from the headache; in other cases the headache gradually subsides. The whole paroxysm lasts from five hours to two or even three days. During the height of the attack of migraine there is generally intolerance of light and sound; and yet, according to E. Souila (*Thèse*, Paris, 1884, No. 35), occasionally there is an intense craving for light, and even for noise.

Although the general features of an attack of migraine conform to the account just given, there are certain symptoms which are occasionally present, and demand more detailed description. In some cases the prodromes are very marked, and include distinct disturbance of a special sense. The sight is the most frequently affected, and next after it the smell. Possibly a peculiar bitter taste in the mouth which seems frequently to precede an attack of migraine, and which is generally referred by most patients to disorder of the stomach, should be noted as among the sensory prodromes. This taste has seemed to me to be closely connected with a peculiar, excessively disagreeable odor of the breath, which in turn appears to be due to the excretion of some sulphuretted compounds. Jewelry about the person may be very distinctly tarnished during an attack.

More usual and more distinctly prodromic is the phenomenon which M. Galezewski has described under the head of *migraine ophthalmica*, or *hemiopia periodica*. The most frequent form of this is an amblyopia, accompanied by vivid scintillations passing zigzag, like the lines of a fortification, over the field of vision. When hemiopia occurs, it may be either monocular or binocular; sometimes it is lateral; in other cases it occupies the superior half of the visual field. In the binocular form a lateral half of the field is attacked. The vision is completely abolished in the affected portion of the field, although the total acuity of vision may remain normal. This sensory disturbance very rarely occurs except in persons who have long suffered from the migraine. In some cases it is preceded by headache, but usually it develops suddenly as the beginning of the paroxysm; occasionally instead of hemiopia a central scotoma is the dominant symptom. Rarely this scotoma merges itself finally in a hemiopia. In rare cases these disturbances of sight are replaced by distinct visions or hallucinations. The olfactory disturbance which ushers in a migraine is generally of a peculiar odor, like that of osmic acid, etc. The auditory prodrome has been variously described as like the sound which is produced when a marine shell is applied to the ear, or as a gurgling similar to that which is heard when water enters the ear during washing. It is stated that in very rare cases a taste comparable to that produced by passing an electric current through the mouth is prodromic of a paroxysm of migraine.

The psychical symptoms which accompany a migraine are usually not severe; but in rare cases they are very marked, affecting especially the emotional nature, causing in one instance profound melancholy and depression, in another, vivacity; in either case there is commonly an excessive irritability. During the attack, according to the measurements of O. Berger, there is a condition of hyperæsthesia of the skin of the face, at least so far as the sense of locality and the electric sensibility are concerned. Certainly in most cases there is no excessive sensibility to pressure, and indeed commonly the pain is more or less distinctly relieved by firm pressure. There is usually no tenderness either during or after the attack at the point of emergence of the nerve from the bone, although in some cases a certain degree of general tenderness of the face is produced by a violent paroxysm. A remarkable but very rare complication of migraine is an aphasia during the height of the attack. Thus, in a case reported (*Gazette des Hôpitaux*, May 17, 1884) by Prof. Charcot, there was habitually complete aphasia for about an hour during the crisis of the paroxysms.

As the affection has come under my notice in this country, vaso-motor disturbance is not usually pronounced; but Eulenburg distinguishes two varieties of migraine which he says are typical. In the one, during the height of the paroxysm, upon the affected side the face is pale, the pupil dilated, the temporal artery hard, and the temperature of the external auditory canal is reduced one to two degrees Fahr. Pressure of the carotid upon the side of the pain now increases the pain, whilst pressure upon the artery on the opposite side of the neck tends to relieve it. Towards the end of the paroxysm the face and ear become red, with a sensation of heat and an absolute rise of the temperature; at the same time there is in some cases a contraction of the pupil. In the second variety of migraine described by Eulenburg there are throughout the paroxysm evidences of vaso-motor depression. Always at the height of the attack the face is red and hot, the conjunctiva injected, and the lachrymal secretion increased. The ear of the affected side is distinctly hotter than its fellow, and the sweat is very abundant at the immediate site of the pain, or sometimes the sweating is unilateral. By compression of the carotid upon the affected side the pain is lessened, but it is increased by

pressure upon the artery of the opposite side. It is affirmed that in some cases the dilatation of the arteries and veins can be detected in the fundus. Towards the close of the attack the face becomes pale.

The existence of these varieties of migraine I have not been able to verify. A very extraordinary phenomenon which is vouched for by the late Dr. Anstie, of London, is that in certain cases of migraine an absolute change in the color of the hair of the eyebrows in the immediate neighborhood of the pain can be seen to occur during the paroxysm, the hair becoming white, but regaining its color after the pain-storm is past: by a succession of these paroxysms the hair is gradually bleached permanently, so that a white lock appears in the eyebrow or even in the head.

The peculiar features of a paroxysm of migraine are usually repeated in the next, the same type of attack being persistent in the one individual.

There have been a number of theories brought forward as explanatory of the attack of migraine. As these theories still remain theories, it is beyond the province of the present work to discuss them. Clinical experience shows, first, that migraine is in some way related to gout; secondly, that in the great majority of cases it is an inherited disorder, which has close relations with other serious neurotic ailments.

Face-Pains.

Probably owing to its great development and to its exposed position, the trigeminal nerve is especially prone to suffer from functional and organic disturbance. The sensory root arises from a nucleus near the floor of the fourth ventricle, and emerges from the pons to enter quickly the Gasserian ganglion, which it leaves in three branches, the ophthalmic, the superior maxillary, and the inferior maxillary. The first of these furnishes branches to the eyeball, to the lachrymal gland, to the mucous membranes of the nose and eyelids, to the skin of the nose, upper eyelid, and forehead, and to the upper part of the hairy scalp. The superior maxillary nerve supplies the integument above and over the maxillary bone, that of the lower eyelid, that of the side of the nose and upper lip, also the upper teeth, the mucous membranes of the nose, of the upper part of the pharynx, of the antrum,

and of the posterior ethmoidal cell; it also sends its branches to the soft palate, the tonsil, the uvula, and the glandular and mucous structures of the roof of the mouth. The inferior maxillary nerve supplies the lower jaw and teeth, the tongue, the mucous membrane of the mouth, and the salivary glands, and sends filaments to the side of the head, the external ear, the external auditory canal, the lower lip, and the lower part of the face.

Much has already been said in this book in regard to pain-storms which affect the trigeminal nerve. In addition to these we must recognize four classes of pain in the nerve:

First. Neuralgic.

Second. Reflex.

Third. Neuritic.

Fourth. Prosopalgic.

By *neuralgia* affecting the trigeminal nerve is meant violent pain occurring in the nerve which is not due to any known organic affection, and which is simply an expression of a general neuralgic temperament, either hereditary or acquired. By most authors the term neuralgic in regard to the trigeminal nerve is given a very much wider significance, so that almost any form of trigeminal pain is spoken of as neuralgic. Trigeminal neuralgia, using the term in its restricted sense, is to be recognized by the coexistence of the neuralgic temperament, and by the absence, between the paroxysms, of the tender points of neuritis. (See page 314.) The neuralgic temperament is itself to be made out by knowing the history of repeated attacks of pain, affecting now this, now that region of the body,—inconstant, shifting, apparently causeless. These pains are the expression of a general neurotic vice.

Reflex trigeminal pains are the result of some more or less permanent irritation situated at a distance from the nerve, and, it may be, in a position having no direct connection with the nerve. These reflex pains usually do not have the history of long continuance that is characteristic of dental and prosopalgic pains. They are also usually much less severe; but they are especially to be recognized by the discovery of a point of irritation, and by the removal of the irritant. They have been noted as following injuries to distant nerves, as due to the irritations produced by lumbricoids or tapeworms, and to over-excitation of the uterine,

ovarian, or other organs of generation, as well as to hæmorrhoids and other rectal diseases.

Trigeminal neuritis is a very frequent affection. In a large proportion of cases it is gouty or rheumatic. The pain is violent, affecting perhaps the whole distribution of the nerves, certainly a large territory, and usually associated with a distinct history of rheumatism in the past or with other evidences of a general disorder in the present. Trigeminal neuritis may be suddenly produced by excessive exposure. It may, under these circumstances, be rheumatic; but it seems to me probable that there is a non-rheumatic neuritis directly caused by exposure. A very important form of trigeminal neuritis is that which is produced by decayed teeth, through the propagation of an inflammation of the pulp along the smaller twigs until the whole nerve is involved. Of similar nature to such neuritis is that which has been described by Prof. Gross as a peculiar neuralgia occurring in the toothless remains of the alveolar processes of old people, and due to the irregular or excessive deposits compressing and irritating the nerves. Dental neuritis is essentially chronic, is associated with horrible suffering, and when once established has little or no tendency to get well, although in the earlier stages it may be arrested by removing the point of irritation,—that is, the affected tooth. A neuritis arising from a decayed tooth is usually confined throughout its course, or at least for many months, to the inferior or superior maxillary branch; and such isolation of a neuritis should always give rise to a suspicion of dental origin.

Trigeminal neuritis is to be distinguished from neuralgia of the nerve by the persistent tenderness felt at the point of emergence of the nerves from the bones of the face. As these points were first pointed out by Dr. F. L. I. Valleix, they are frequently spoken of as Valleix's points.

In the ophthalmic branch, the most important point is the supra-orbital foramen. Less commonly to be recognized are the palpebral points of the upper eyelid, the nasal on the nose, where the ethmoidal nerve emerges from the nasal cartilage, the inner angle of the eye, corresponding to the supra-trochlear nerve, and the parietal prominence. In the superior maxillary branch, the most important point is over the infra-orbital foramen; next in order is that in the upper lip, then those in the gums or in the

alveolar processes of the upper jaw. In the inferior maxillary branch, the point on the chin is the most frequent, next is one in front of the ear, while very inconstant and rarer are points on the lower lip, on the side of the tongue, and on the alveolar processes of the lower jaw.

Under the term *prosopalgia* I include all trigeminal pains which are neither neuralgic, reflex, nor neuritic. The suffering in prosopalgia is apt to be intolerable; pains of the most furious character shoot with lightning-like rapidity along the course of the nerves, follow one another in incessant flashes for a few seconds or minutes, and then abruptly cease. In many cases these pains are accompanied by furious clonic and tonic contractions of the muscles of the side of the face, giving rise to the congeries of symptoms known as *tic-douloureux*. In some cases paroxysms occur only a few times a day, but more frequently they repeat themselves at short intervals. The lower jaw and cheek are probably the most frequent seats of the pain; somewhat more rarely are the branches of the upper maxillary and even of the ophthalmic nerves affected. Only in exceptional cases do the mucous membranes suffer; but frightful burning, shooting, stinging, darting pains may be felt in the mouth, and become excessively severe as they run through the tongue. Often, impelled by an irresistible impulse to do something, or perhaps led by a slight feeling of relief, the patient, during the paroxysms, incessantly rubs the affected part with the hand, either naked or armed with a handkerchief, and it is not uncommon under these circumstances to see the cheek bare of skin from incessant rubbing. These severer forms of prosopalgia are capable of being arranged in two groups,—those in which a lesion can and those in which it cannot be demonstrated. I have seen a violent trigeminal *anæsthesia dolorosa* immediately follow an apoplexy, and due, without reasonable doubt, to the involvement of the sensory nucleus or fibres of the trigeminal nerve in the pons. Cruveilhier found a cancerous growth attacking the nerve-sheath in a case of obstinate prosopalgia. Laveran noted a fibrous degeneration of the Gasserian ganglion; Lippic, caries of the bone immediately below the outgoing nerve-root; and syphilitic, cystic, and gliomatous tumors have been found in the neighborhood of the Gasserian ganglion. A progressive lesion running a not too rapid course and involving

the sensory roots or nucleus of the trigeminal nerve, either before or after they enter the Gasserian ganglion, is often the cause of an obscure prosopalgia. To cases of prosopalgia without obvious lesion Prof. Trousseau gave the name of epileptiform neuralgia, because of his belief that the affection is related to epilepsy. This opinion was founded on his having seen disorder of the intelligence in some cases, and having noted in others that the attacks were ushered in by vertigo or other aura-like sensation, whilst one or two epileptics suffering from violent prosopalgia had come under his notice. Obstinate, apparently causeless prosopalgia occurs especially in old people: it is probable that it is the result of atrophic or nutritive degenerations of the nuclei or of the root of the nerve, such degenerations often being caused by alterations of the walls of the blood-vessels. I can see no sufficient reason for believing that a paroxysm of tic-douloureux ever represents one of idiopathic epilepsy.

CHAPTER VIII.

DISTURBANCES OF THE SPECIAL SENSES.

HEARING.

For testing the hearing the neurologist usually relies upon the ordinary watch or a tuning-fork. In many cases the acuteness of audition is to be determined by comparing the two ears of the patient. If both sides are equally affected, it becomes necessary to compare the hearing of the individual with that of another person. The varying loudness of the ticking of watches and the great natural differences in the acuteness of hearing in different individuals render an accurate estimation of the amount of a double deafness difficult. There is an instrument known as the tonometer, used by otologists, which is supposed to give always a sound of a definite intensity; but it is probable that even this instrument as made by different instrument-makers varies in the intensity of its sound. Moreover, I know of no studies in which the range of normal audition has been determined: so that the ordinary practitioner is usually forced to take his own hearing as the normal standard.

In examining a case of deafness the neurologist must first determine whether the existing deafness is due to disease of the middle ear or is neurotic. In doing this it is customary to employ the tuning-fork. If the deafness be unilateral and be due to obstructive disease, either of the middle or of the internal ear, when a tuning-fork in vibration is placed upon the vertex the noise is heard much more intensely in the deaf ear. Under such circumstances the neurologist knows that the loss of hearing is, at least in part, due to wax in the external meatus, stoppage of the Eustachian tubes, or other obstructive disease. Unfortunately, in certain middle-ear diseases, such as anchylosis of the bones, the tuning-fork is not heard more loudly on the deaf side. As, however, these cases are comparatively rare, it is a general rule that when the tuning-fork is not heard more loudly on the deaf side the deafness is probably due to a lesion of the nerve itself or of its centre. We have no way of determining with certainty whether

such loss of hearing is due to a lesion in the labyrinth, in the trunk of the nerve, or in the nerve-centres: labyrinthine disease is, however, generally connected with severe giddiness, whilst in disease of the nerve or of its centre giddiness, if it exist, is not severe.

The eighth, or auditory, nerve has its principal nucleus in the floor of the fourth ventricle, close to the nuclei of the vagus, the glosso-pharyngeal, and the hypoglossal nerves. From this nucleus the inferior root arises. The second, outer, superior, or minor auditory nucleus lies between the inner nucleus and the restiform body, and gives origin to the superior root of the nerve. The roots pass obliquely outward and unite into a single trunk, which appears at the lower edge of the pons on the outer side of, and close to, the facial nerve. After leaving the medulla oblongata the nerve is directed outward, in company with the facial nerve, to the internal auditory meatus.

Deafness from disease of the auditory nuclei is very rare. Peripheral neurotic deafness is much more common. The auditory nerve is liable to be pressed upon by syphilitic, tubercular, or other exudations at the base of the brain, and is especially exposed to paralysis from disease of the mastoid processes of the temporal bone. It may therefore be laid down as a general diagnostic rule, the exceptions to which are very rare, that a nervous deafness not associated with marked giddiness is dependent upon a lesion of the nerve-trunk.

Hyperæsthesia of the auditory nerve produces a loss of hearing which is characterized by excessive susceptibility to sounds. The normal stimuli of the nerve produce pain rather than normal functional excitement, so that, although unable to perceive minute differences in sounds, the patient suffers acutely from loud noises.

SIGHT.

In discussing the relation of the eye to diseases of the nervous system, I shall take up first its movements, next alterations of the pupils, and then internal conditions.

MOVEMENTS OF THE EYE.

Strabismus.—When one or more of the eye-muscles are paralyzed or excessively contracted, the axis of the eye is drawn

out of its normal direction,—in the one case by the sound muscles, which are no longer controlled by their normal antagonists; in the other case by the overacting muscle or muscles. When from any cause the axes of the eyes do not correspond, the patient is said to suffer from strabismus, or squint. A strabismus may be either paralytic or concomitant. Paralytic strabismus is almost invariably the result of disease of the nervous system; concomitant strabismus is the outcome* of an ocular defect. In the hypermetropic eye the squint is usually convergent; in the myopic or near-sighted eye it is usually divergent.

In any individual case the first point in the diagnosis is to determine whether the existing squint is paralytic or concomitant. The diagnosis as to which muscle is affected is to be made by studying,—first, the movements of the eye; secondly, the so-called secondary deviation; thirdly, the absence or presence of double vision, and, as part of this, the false projection of the field of vision of the paralyzed eye.

First. With the patient sitting before him, the practitioner holds up the finger or the point of a pencil and requires the subject to follow its movements with the eye, the head being kept still. Under these circumstances, if the squint be paralytic the movements of the ball will be found to be restricted on the side of the paralyzed muscle. This test seems a very simple one, but, owing to the complicated relations of the ocular muscles, in some cases judgment is difficult.

Secondly. It is well known that one eye habitually follows the movements of the other eye, so that if the left eye be directed towards an object situated on the extreme left the right eye follows it even though covered. The movements of the second eye depend upon the fact that when a nerve-centre is called upon to move one eye it sends an equal and parallel amount of force to the other eye: thus, if the external rectus and associated muscles be called upon in one eye, the internal rectus and associated muscles in the other eye receive an equal amount of nerve-force. When a partially paralyzed muscle is required to turn the eye in a certain direction up to a certain amount, it is plain that a much greater

* The explanation of the connection between ocular defects and strabismus belongs to works on diseases of the eye, as it is a purely local matter.

exertion on the part of the nerve-centre controlling the movement will be required to produce the desired movement than would be necessary if the muscle were not paralyzed. The amount of nerve-force discharged from the centres being equal in the two eyes, it follows under the circumstances just spoken of that the muscle of the normal eye will receive a greater amount of nerve-force than is necessary to carry the eyeball over the required distance: the eye will, therefore, be drawn beyond the object which is to be looked at. If, after such movement, the normal eye be directed towards the object, the ball will have to return towards its central position,—the amount of return being exactly the excess of movement first produced. This return is the measure of the "*secondary deviation*,"—the secondary deviation being the distance which the sound eye has been drawn beyond the position which it normally should have assumed. *Primary deviation* is the deviation of the eye whose muscle is paralyzed, from its normal position, when the object is looked at with the sound eye.

To make this matter clearer, let us suppose that x represents the amount of force required to move the normal left eye a given distance towards the left, and that the muscles of the left eye become so far paralyzed that $2x$ is required to execute the necessary movement. When such movement is executed, the sound eye will receive $2x$ instead of x nerve-force, and will therefore be drawn twice as far from its central position as is required. The secondary deviation will therefore be the amount of movement which is produced by the expenditure of x force in the eye. In order to make clear the method of testing the presence of secondary deviation, let us suppose the patient has a defect in the left eye. The right or sound eye is covered with the hand or a piece of paper, whilst the left or affected eye is directed towards an object situated at the distance of a few feet. When the left eye has been focussed upon such an object, the patient is told to look at it with the right eye, and the cover hastily removed: the right or sound eye will now be seen to move in a direction opposite to that towards which the paralyzed muscle draws the ball. Thus, if it be the external rectus of the left eye that is affected, the right eye when uncovered will move backward towards the right, because its internal rectus has originally received an excess of nerve-force and drawn it too far towards the left.

Thirdly. If by pressure upon the ball the axis of one eye is distorted, two objects are seen, because the visual fields of the eyes no longer correspond. Double vision is equally produced when the distortion of the axes is the result of a paralysis and when it is caused by an overaction of one or more muscles. When the lesion of the muscle and consequent distortion of the axes has been very gradually developed, the patient as gradually loses the perception of one of the two images, or, in other words, habitually sees with only one eye. The concomitant squints due to defects of the eye are very slowly developed, so that the second image is habitually lost and the patient has conscious vision only of the single object. On the other hand, paralytic squints are usually suddenly or rapidly developed, and the habit of not heeding one image has not time to form itself, so that double vision results. For these reasons we are able to frame the diagnostic rule that *whilst a paralytic squint is almost invariably accompanied by double vision, a concomitant squint is rarely so accompanied.* In most of the cases in which the second image has been lost through habit it can be developed by placing a colored glass before the sound eye, so as to tint one of the images. When the images are so near together that they overlap, the result is a blurred image, which by the colored glass is resolved into two.

Double vision may be crossed or simple. *Crossed diplopia* occurs in cases of divergent squint: the image seen by the left eye is to the right of that seen by the right eye. In other words, the image seen by the left eye is carried beyond or crossed over that seen by the right eye. *Simple* or *homonymous diplopia* exists in convergent squint. In it the image seen by the left eye is to the left of the other image. A great aid to the memory in regard to this diplopia is afforded by remembering the rule laid down by Gowers in his lectures,—namely, that when the prolonged axes of the eyes would cross, the images are not crossed; whilst when the prolonged axes would not cross, the images are crossed. In other words, convergent squint causes simple diplopia, divergent squint causes crossed diplopia.

In long-existing cases of paralytic squint, secondary contractures of the non-paralyzed muscle may give rise to diplopia of peculiar character. For the discussion of these finer points the reader is referred to works upon the eye.

The *false projection of the visual field* is a subject which seems at first very abstruse, but which may readily be simplified. According to my thinking, it really is neither more nor less than diplopia, although Gowers speaks of it as though it were a distinct symptom. We judge of the positions of objects in regard to our own body by their relations to our visual fields. This relation depends upon the position of the head and of the eyes, so that the degree of contraction of the muscles of the head and of the eyes—*i.e.*, the amount of nerve-force which is given off to them—unconsciously becomes to our consciousness a measure of the position of objects. If the muscles of the eye are at rest, the ball is in mid-position, and we know that an object upon which we are focussing the eye, or, in other words, at which we are intently looking, is directly in front of the face; that is, we know that a line drawn from the centre of the object at which we are looking to the centre of the field of vision—*i.e.*, to the visual centre of the retina—would be at right angles to the face. If the eye is now focussed upon an object to the left, the degree of movement of the eye—that is, the amount of nerve-force given to the muscles of the eye—is the basis of the unconscious judgment which leads to the recognition of the position of the object relative to our own bodies. Experience has shown us that the expenditure of x force on the appropriate muscles in turning the eyes to the left is required if the body looked at is situated at a certain angle in regard to ourselves. If the object looked at is sufficiently close for us to lay our hands upon it, we unconsciously throw into the nerves of the arm force which in amount and direction corresponds to that given to the eye-muscle, so that the hand is placed directly upon the object seen. If the eye-muscle is partially paralyzed, and an increased amount of nerve-force is required to produce the necessary contractions of the eye-muscles in looking towards the object, the lower nervous system throws into the hand, which is put forth to touch the object, force which in amount and direction corresponds to the abnormally large force required to move the weakened eye-muscle. The hand is therefore projected too far to one side of or beyond the object, according as this or that ocular muscle is paralyzed. The false direction of the arm will accord with the direction of the secondary deviation of the sound eye; that is, if the normal eye goes too far to the left the arm will

go too far to the left, etc. The discord which thus arises between the face of things as seen and as felt often produces vertigo or giddiness: hence the so-called ocular vertigo. (See article on Vertigo.) In some cases the nerve-centres finally learn to accommodate themselves to the altered circumstances. In other cases the patient may try to avoid the vertigo by holding his head in such a position that the affected muscles are not called into action, or by closing the affected eye, either with the hand or, more generally, by a contracture of the orbicular muscle. As pointed out by Gowers, the affected eye is always the one closed, because, although closing either eye would remove the diplopia, only closure of the affected eye removes the vertigo or uncertainty, by removing the discord between the visual and other sensations.

This scientific explanation of the false projection of the visual field is, of course, interesting, but after all it amounts only to this, that the patient sees the image of the object looked at in a false position, and that he naturally puts his hand forward to seize the image at the position in which he sees it. Thus, if in a case of diplopia two distinct inkstands are seen, although only one exists, and the secondary inkstand is perceived to the left of the true one, when the sound eye is closed the patient sees only the secondary or left image. If now, with the sound eye closed, he should attempt to take hold of the inkstand, he would of course seize, not the inkstand proper, but the secondary image which alone he sees.

Oculo-motor Palsy.—A paralytic divergent squint is due to palsy of the third, or oculo-motor, nerve, which supplies all the muscles of the orbit except the superior oblique and external rectus. As it is possible for certain fibres of the oculo-motor nerve to be paralyzed and the remainder to preserve their normal activity, one of the muscles supplied by the eye may be alone affected. This necessitates a brief consideration of the varieties of strabismus connected with each muscle.

When the internal rectus is paralyzed, there is defect of the inward movement of the eye, and crossed diplopia, the false image being oblique above and below the horizontal plane; when the superior rectus alone is paralyzed, the movement upward and outward is affected: a certain amount of upward and inward movement is preserved in the ball, because the inferior oblique muscle habitually acts with the superior rectus in causing those

movements: double vision occurs on looking upward, the false image being above the true. When the inferior rectus muscle is paralyzed, there is loss of the downward and outward movements: the secondary deviation is produced by the opposite rectus moving the ball too much downward and outward, so that the return movement is upward and inward. This form of strabismus is apt to be confounded with that which results from paralysis of the superior oblique (fourth nerve: see p. 325). Paralysis of the inferior oblique muscle causes defect of the upward movements: the secondary deviation is produced by excessive action of the inferior oblique and internal rectus muscles of the sound eye, so that the ball is carried too much upward and inward and the return movement is outward and downward: double vision occurs with the false image oblique and situated above the true, the obliquity being greatest in looking outward, the defect in height in looking inward.

The *third*, or *oculo-motor*, nerve emerges from the inner side of the crus cerebri close to the upper border of the pons, and extends outward and upward between the posterior cerebral and superior cerebellar arteries to the outer side of the posterior clinoid process, a little anterior to which it penetrates the dura mater close to the outer boundary of the cavernous sinus, and passes through the sphenoidal fissure. Its deep root is situated in the gray matter of the floor of the Sylvian aqueduct, in the region of the superior corpora quadrigemina, just above the nucleus of the fourth, or trochlear, nerve. The fibres pass forward from the nucleus through the tegmentum and the tegmental nucleus, and partly through the substantia nigra to the point of emergence. Owing to the position of its nucleus, the oculo-motor nerve is liable to be paralyzed by lesions of the crus cerebri. Under these circumstances the symptoms of the oculo-motor palsy are upon the side of the body opposite to the hemiplegia or hemianæsthesia.

As the oculo-motor nerve supplies not only the muscles of the eyeball, but also the elevator of the upper eyelid, the levator palpebræ, its complete palsy is followed by ptosis, paralysis of accommodation, and dilatation of the pupil, as well as by paralysis of the muscles of the ball. The discussion of the relations of the oculo-motor nerve to the pupil will be found under the head of Pupil.

Partial paralysis of the oculo-motor nerve has a significance

similar to the same phenomenon in the abducens, with the exception, at least in my experience, that functional palsy is more rare. Owing to its prolonged course at the base of the skull, it is especially liable to be pressed upon by syphilitic or tubercular exudations. An acute oculo-motor palsy which is not rheumatic is in the adult generally syphilitic, and in the child usually tubercular.

According to the statistics of Eulenburg, the oculo-motor nerve is more frequently affected in locomotor ataxia than are the other ocular nerves. Under these circumstances the paralysis is nearly always partial; very rarely is there ptosis, usually the muscles and the pupils alone being affected. On the contrary, it is not rare in syphilis for ptosis to exist without marked disorder of the pupil or of the muscles of the eyeball.

Paralysis of Trochlear Nerve.—Loss of movement of the eye downward and inward, with the convergent strabismus most marked when the patient looks down, is due to the paralysis of the superior oblique muscle, which is supplied by the fourth, trochlear, or pathetic nerve. Care may be necessary to distinguish paralysis of the superior oblique muscle from that of the inferior rectus, in which the loss of movement is downward and outward: the distinction is readily made by paying attention to the position of the images in double vision. In paralysis of the inferior rectus the diplopia is crossed, and the false image is placed below the true and is oblique. The greater the attempted movement, the lower it is situated and the more oblique it is. In paralysis of the superior oblique muscle the diplopia is simple, and, the false image being lateral, the distance between the two images is greatest in the middle line, and lessens when the object is moved inward or outward. The diplopia occurs especially when the patient looks downward, and often gives much trouble in going down-stairs, the patient being unable to distinguish the false from the true image of the descending flight. The trochlear or pathetic nerve has its superficial origin just below the corpora quadrigemina, and is directed at first outward across the superior peduncle of the cerebellum, and then turns forward around the outer side of the crus cerebri, between the posterior and superior cerebellar arteries. Its nucleus is situated between the superior and inferior quadrigeminal bodies, immediately below that of the third nerve. A

partial decussation of this nerve is said to occur in the anterior medullary velum at the commencement of the aqueduct. Paralytic affections of the trochlear nerve are very rare, but may occur under circumstances similar to those of the oculo-motor nerves.

Paralysis of Abducens Nerve.—Paralytic convergent squint is due to paralysis of the external rectus muscle. The outward movements of the eye are restricted; the diplopia is simple. The two images are parallel and on the same plane when on the horizontal plane of the centre of vision, but they are usually more or less oblique and on a different plane when they are situated above or below the horizontal visual plane,—*i.e.*, when the eye has to be moved upward or downward.

The external rectus is supplied by the abducens, or sixth cranial, nerve. This nerve arises in the groove between the pons and the medulla oblongata, immediately external to the upper end of the pyramid, and, going outward, lies close to the floor of the cavernous sinus, in contact with the outer side of the internal carotid artery; finally it passes through the sphenoidal fissure to the muscle. Its deep root is situated in the floor of the fourth ventricle; a few fibres are believed to pass upward and across the raphé to join the third nerve of the opposite side. In this is to be found the explanation of a few recorded cases in which atrophy of the nucleus of the sixth, or abducens, nerve has been followed by paralysis of the internal rectus of one side and the external rectus of the other side,—*i.e.*, by a convergent squint on the side of the lesion and a divergent squint on the opposite side.

Partial paralysis of the external rectus not rarely occurs from simple neurasthenia, especially when the cerebral exhaustion is largely manifested by disorder of vision. In such cases, and also in cases of locomotor ataxia with strabismus, double vision from disorder of the optic axis may occur although the strabismus is not sufficient to be easily noticeable. Paralysis of the external rectus, as of other eye-muscles, may be rheumatic. In such cases there is usually a history of exposure, pain in the eyeball or its neighborhood, swelling and pain in the face, and various evidences of a rheumatic diathesis. Attempts to move the eyeball also cause pain.

A complete, non-rheumatic paralysis of the abducens nerve always depends upon an organic lesion either of the nerve itself

or of its centre. In cases of tubercular or syphilitic basal meningitis the nerve is very apt to be pressed upon; and it may be laid down as a sufficiently accurate rule that *acute, complete, non-rheumatic paralysis of the external rectus in the child is tubercular or syphilitic, in the adult syphilitic, unless due to cerebro-spinal meningitis.*

In certain cases of poliomyelitis the nucleus of the abducens undergoes wasting, and the muscle suffers from atrophic palsy. A convergent strabismus from paralysis of the sixth nerve is also an occasional phenomenon in posterior sclerosis. It was present in six of the twenty-five cases of locomotor ataxia with strabismus recorded by Eulenburg. This form of convergence is, like all other forms of strabismus due to locomotor ataxia, apt to occur at first in paroxysms—*i.e.*, to come and go—and to be associated with giddiness. It may be a very early phenomenon, preceding the more pronounced symptoms of posterior sclerosis. When the sclerosis commences high up, strabismus, atrophy of the optic disk, and fulgurant pains in the distribution of the fifth nerve may long precede the usual symptoms of locomotor ataxia. (See Clozier, *Recueil d'Ophthalmologie*, 1880, ii.; also Galezowski, *ibid.*, 1884, vi. 334.) Paralysis of eye-muscles, with or without alterations of the pupils, is somewhat common in multiple sclerosis, having been noted in thirteen of the fifty cases observed by R. Gnauck (*Neurolog. Centralblatt*, 1884, iii. 314). In some cases of sclerosis nystagmus is present.

Ophthalmoplegia Interna.—Under the name of ophthalmoplegia interna Mr. Jonathan Hutchinson describes (*Med.-Chir. Trans.*, vol. lxi. p. 215) an affection of the eye which he believes to be the result of paralysis of the ciliary ganglion. In this ganglion the fibres of the oculo-motor nerve meet with those nerve-fibres which, originally springing from the cilio-spinal axis of the cervical cord, pass upward through the superior cervical sympathetic ganglion to the brain. From the ciliary ganglion the conjoined sympathetic and oculo-motor fibres pass outward to supply all muscular fibres within the eyeball. Destruction of the ciliary ganglion is consequently followed by *iridoplegia*, or paralysis of the iris, both as to the circular and the radiating fibres, and by *cycloplegia*, or paralysis of the ciliary muscle. I have never recognized a case of ophthalmoplegia interna, and I think there is difficulty in its positive diagnosis. Unfortunately, I have not

been able to find in any of the various papers of Mr. Hutchinson upon this subject a solution of the difficulties. Either the oculo-motor nerve, which supplies the circular fibres of the pupil, or the sympathetic nerve, which supplies the radiating fibres, may be separately paralyzed, or the two may be conjointly paralyzed. In either case the pupil is immovable to light or other influence. When the oculo-motor nerve alone is paralyzed, the pupil is dilated. When the sympathetic nerve alone is affected, the pupil is contracted. When both nerves are paralyzed, the pupil must be of medium size. The degree of dilatation in the oculo-motor palsy varies, however, greatly, and I strongly suspect that some of the recorded cases of ophthalmoplegia interna were simply instances of oculo-motor paralysis.

Ophthalmoplegia Externa.—Ophthalmoplegia externa is a name applied by Mr. Hutchinson (*Med.-Chir. Trans.*, vol. lxii.) to those cases in which all the external muscles of the two eyes are more or less completely paralyzed. Long before the name was given by Mr. Hutchinson the condition was described by Von Graefe as *ophthalmoplegia progressiva*. If the palsy is nearly complete, there is marked drooping of the upper lid, with complete immobility of the eyeballs, giving rise to a very peculiar expression of the face. Usually the internal muscles of the eye are also implicated, but occasionally, according to Mr. Hutchinson, they escape. The causes of the affection are various. Paralysis of all the muscles of a single eye is in the majority of cases due to pressure upon the nerves at the base of the brain: in adults such pressure is usually caused by syphilitic exudation. A double ocular paralysis may evidently be caused by a syphilitic or other growth of such size and situation as to press upon the nerves of both eyes. Ophthalmoplegia externa may, however, be of centric origin. A case is reported by Mr. Hutchinson in which, at the autopsy, was found degeneration of the nuclei of the affected nerves apparently identical with that which occurs in progressive muscular atrophy. Dr. Thomas Buzzard (*Brain*, vol. v. p. 34) has recorded the case of a syphilitic woman in whom ophthalmoplegia externa coexisted with symptoms of locomotor ataxia, and in whom there was found, after death, degeneration of the nuclei of the ocular muscles and of some portion of the gray matter of the cord, and also pronounced posterior scle-

rosis. According to Dr. Edward Nettleship (*Diseases of the Eye*, Philadelphia, 1883, p. 392), in young adults a functional ophthalmoplegia externa sometimes develops, with symptoms which come on quickly and pass off completely; in some cases there are repeated attacks.

Associated Paralysis of the Eye.*—In certain cases there is paralysis of the eye-muscles which are associated with one another in their movements. Thus, the vertical movement may be lost in each eye. In such a case the obvious explanation offers itself that the nuclei of the affected nerves are symmetrically diseased. When, however, the associated paralysis involves the lateral movements of the eye, the muscles which are implicated are not supplied by the same nerves. Thus, in the left eye there would be loss of power in the external rectus muscle, whilst in the right eye the internal rectus would be affected. In such a case a post-mortem examination by M. Féréol demonstrated the lesion to be situated on the level of the eminentia teres and to affect the nucleus of the sixth pair, which is joined by a band of fibres with the oculo-motor nucleus of the other side. (See page 87.) Associated paralysis of the eye is asserted to be frequent in multiple cerebro-spinal sclerosis (Parinaud, *Progrès Médical*, xii. 641); but as that disease occurs in this country it must be a very rare complication, since I have never seen it.

Conjugated Deviation of Head and Eyes.

As long ago as 1834, M. Andral, in the third edition of his *Clinical Medicine*, called attention to a symptom occasionally occurring in apoplexy which has recently been much commented upon.† In ordinary hemiplegia the tendency of the head is to fall passively towards the paralyzed side, but in certain cases the head

* For an elaborate paper on this subject by H. Parinaud, see *Archives de Neurologie*, vol. v. p. 145.

† The reader desirous of following up this subject will find most of the references up to the date of its publication in the monograph by Dr. J. L. Prevost, *De la Déviation conjugée des Yeux, et de la Rotation de la Tête dans certains Cas d'Hémiplégie.* Since that date the papers of Dr. Broadbent (*London Lancet*, vol. ii., 1879, p. 861), Dr. Landouzy (*Progrès Médical*, 1879, vol. vii. p. 957), and Dr. Bechterew (*St. Petersburger Medicin. Wochenschrift*, March 14, 1881) are the most important on the subject.

is drawn forcibly from the paralyzed extremities, and the eyes with their axes parallel are also forcibly directed towards the sound limbs. This so-called conjugated deviation of the head and eyes varies in intensity. The spasm of the affected muscles is sometimes so intense that it is almost impossible to restore the head to its normal position. Usually, however, the head can be put back, but returns to its abnormal posture the instant the force is withdrawn. In milder cases it may be very easy to restore the head, and no immediate movement may follow letting it go, but slowly the head returns to its original position. When consciousness coexists with this symptom, except in very mild cases, the patient has no control over the head and eyes.

Conjugated deviation of the head and eyes is much more apt to develop when the hemiplegia comes on suddenly; and in severe cases of apoplexy, when all the limbs are so flaccid that it may be difficult to discover the existence of the local palsy, the diagnosis of cerebral hemorrhage may occasionally be made out by noting the distorted position of the head and eyes. It is true that in the beginning of an epileptic convulsion drawing of the head and eyes may occur, but it lasts only for a minute or two. Jacksonian epilepsy often begins with conjugated deviation, and when the convulsive seizures follow one another very closely the spasm of the neck- and eye-muscles may be persistent (Bechterew).

The pupil may be drawn into the canthus of the eye. Ordinarily the balls are entirely quiet; but marked nystagmus is not very rare. Conjugated deviation is commonly fugitive. It may cease immediately after the development of the full apoplectic symptoms, but it commonly disappears in a few hours, or at most in a few days. In fatal cases it is not uncommon to see it cease just before death. This fugitive character is not, however, universal, for in some recorded cases the distortion has persisted for a month or more, or even for a whole year. Occasionally during an attack of apoplexy the deviation of the eyes and head may return after having disappeared, or the symptom may be first developed in the midst of the apoplectic storm: under these circumstances it marks the renewal of the hemorrhage.

Although in the majority of instances the face and eyes are directed away from the paralyzed side and towards the lesion, there are exceptional cases which are not at present readily explained.

Among the cases collected by Prevost there are three in which the direction of the head was towards the paralyzed side. In each of these cases the lesion was in the peduncle of either the cerebrum or the cerebellum. On the other hand, in a similar case reported by Dr. Bernhart (*Virchow's Archiv*, vol. lxix.) the lesion was a very large meningeal hemorrhage, and in the reported case by Dr. Nothnagel (*Diagnos. der Gehirnkrankheiten*, p. 682) it was a softening in the cortical motor zone. These exceptional cases would seem to show that the law formulated by Vulpian and Prevost, that in lesions of the hemisphere the head is drawn towards the lesion and away from the paralysis, whilst in lesions of the mesencephalon it is drawn away from the lesion and towards the paralysis, has exceptions. According to Dr. Landouzy, when there are unilateral convulsions with conjugated deviation, if the head and eyes look towards the convulsed extremities there is an irritative lesion of the hemisphere, but if the head is turned away from the convulsed limbs the irritative lesion is in the mesencephalon.

INTERNAL OCULAR CONDITIONS.

The internal conditions of the eye which it is necessary to study in their relations to the nervous system are—first, alterations of the optic disk, or end of the optic nerve; secondly, alterations in the retinal power of receiving impression.

ABNORMALITIES OF THE DISK.

Five years after the discovery of the ophthalmoscope Von Graefe called attention to the marked alterations in the intraocular ends of the optic nerve which frequently occur in intracranial disease. Of these alterations he described two main varieties,—the one in which there is intense swelling of the intraocular end of the nerve, and the other in which there is a dull suffusion of the disk. To the first of these he gave the name of *stasis papillæ*. It is now generally known as "choked disk." The second he designated as descending neuritis.

In choked disk the end of the optic nerve projects into the eye as a small protuberance or umbonation. Its height may be equal to its own diameter. Through its œdematous and opaque nerve-fibres run the tortuous, enlarged, and sometimes newly-

formed ocular vessels, which hide the arteries and allow only the branches of the tortuous and dilated retinal veins to be seen sloping down from the swollen papilla to their normal level in the retina. In descending neuritis the disk is slightly swollen, dull red, with an opacity of its nerve-fibres which completely hides its normal boundaries. The tortuous veins and arteries are often diminished in size.*

Typical cases of choked disk and descending neuritis, seen at the height of the disorders, are said to be distinguishable, but usually they shade off so imperceptibly one into the other, and terminate in atrophies which present so absolutely the same appearance, that it is impossible to distinguish between them.

Choked disk in most cases develops slowly, requiring from a few days to a month to attain its maximum. After this it may remain unchanged for a year, or even more. Ordinarily, however, atrophy begins in the course of a few months. It is remarkable how nearly perfect vision may be even when the disk is enormously swollen; but when atrophy is fairly established almost invariably the amblyopia becomes apparent. In the diagnosis of neuritis some care on the part of the beginner is necessary not to mistake for descending neuritis the neuro-retinitis due to eye-strain from incorrect visual defects or from local congestion from other causes.

Von Graefe explained the choked disk by supposing that the returned blood in the cerebral sinuses is dammed up by the gross lesion of the brain and causes an impeded circulation with increased blood-pressure in the ophthalmic vein and its branches,—the effect of this damming back being increased by the rigid tissue of the lamina cribrosa acting like a multiplier and increasing the pressure at the head of the nerve. This theory of Von Graefe, however ingenious, has been abandoned by ophthalmologists in favor of one or the other of two theories. The first and the least probable of these is that the choked disk is due to paralysis of the vaso-motor nerves connected with the blood-supply in the

* The author acknowledges his especial indebtedness to the able articles by Prof. William Norris ("Medical Ophthalmology," in the 5th vol. of the *System of Practical Medicine, by American Authors*, Philadelphia, 1886) and Prof. E. C. Seguin ("Hemianopsia," *Journal of Nervous and Mental Diseases*, January, 1886).

pupil. The second is the so-called lymph-space theory. As shown by the anatomical researches of Schwalbe, Retzius, and later anatomists, the sheaths of the optic nerves communicate freely with the pia mater and the arachnoid spaces of the brain, which are in turn portions of the lymphatic system of the cerebrum. When, therefore, owing to a gross lesion, the lymphatics of the brain become choked, there is an excessive pressure upon all the lymphatic spaces, which especially expresses itself in the lymph-spaces of the optic disk, a free, unsupported portion of the system.

The latter view is confirmed by experiments upon the lower animals and upon the human cadaver, and also by a large number of autopsies, which have shown that choking of the disk has been accompanied by dilatation of the outer sheath of the nerve by lymph, pus, or blood which has found its way down from the cranial cavity. It has also been proved that growths in contact with the distal end of the nerves may produce choked disk by causing a local accumulation of fluid. The fact that in certain cases of choked disk no distinct lymph-lesions have been found after death is explained by the supposition that such dilatation of the vessels has disappeared in the secondary inflammations and degenerations which have been set up.

Whatever theories we may adopt to explain the production of choked disk and of optic neuritis, clinical experience shows that double choked disk is generally dependent upon brain-tumor, abscess, meningitis, or other gross lesion, and that descending neuritis may be produced by a basal meningitis. For practical purposes we may consider the two lesions as identical, although if in any case a typical descending neuritis is found the probabilities are in favor of the existence of a basal meningitis rather than of any other form of gross brain-lesion. A basal meningitis may, however, produce a typical choked disk. One disk may occasionally be affected earlier than the other. Under these circumstances the lesion is usually on the side of the nerve first attacked. A double optic neuritis sometimes occurs in advanced Bright's disease. It is probable that in such cases there is always serous effusion into the subarachnoid space of the brain, or, in other words, that there is gross lesion of the brain. It has already been stated that tumors and other diseases far back

in the orbit may produce a choked disk, such choked disk being usually unilateral. If a choked disk be unilateral it is probably due to local disease: nevertheless it may be the outcome of a coarse brain-lesion.

Although in a great majority of cases optic neuritis is the result of gross cerebral disease, it may be a primary affection, following typhus, typhoid, scarlet fever, measles, variola, and other constitutional diseases. There is also a form of it which is rheumatic, or at least is directly produced by exposure. It may also develop without discoverable cause, as in a case which I watched for many months without being able to detect any evidences of rheumatism or of cerebral or kidney disease. Sudden blindness, with neuritis, is said to have been produced by the arrest of menstruation by exposure, and to have been recovered from by the restoration of the flow. For details of this and other cases the reader is referred to the paper by Dr. H. F. Hansell (*Medical News*, vol. xlix. p. 144). This idiopathic or rheumatic optic neuritis is frequently monocular.

The absence of choked disk does not prove the non-existence of gross brain-lesion. In a case under my care the eyes were examined two days before death by one of our most eminent ophthalmologists, and the nerve pronounced absolutely healthy: yet a large tumor of the frontal lobe was found at the autopsy. This is in accord with the general experience that lesions of the frontal lobe are especially apt not to give rise to changes in the optic papilla. Gowers believes that disk-changes occur in eighty per cent. of all cases of cerebral tumors. In a series of eighty-eight cases quoted by Norris, the disk was altered in ninety-three per cent. Hughlings Jackson calls attention to the fact that optic neuritis is essentially a transient symptom, which often occurs early in the disease, but may be developed only in the last weeks before death. A slowly-growing tumor or exudation which does not affect the optic chiasm or the optic nerves may remain for months or years without causing an optic neuritis, because it does not materially increase the pressure upon the brain or set up inflammation of the lymphatics. When the alterations of the brain are rapidly progressive, or when they are accompanied by much irritation, infiltration of its nerve and sheath with lymph or inflammatory products must rapidly ensue.

When a cerebral tumor presses upon the intra-cranial portion of the optic nerves, or when the chiasm is compressed and atrophied by the protuberant and bulging floor of the third ventricle, as in cases reported by Foerster, optic atrophy may occur without precedent choked disk.

Atrophy of the Optic Papilla.

Atrophy of the optic papilla may be produced by choked disk, by certain diseases of the cerebro-spinal axis, especially sclerosis, and by affections of the eye itself.

After a choked disk has continued for a greater or less length of time, the swelling begins to subside and the reddish tint to pass slowly into a dull, opaque, grayish-white color. The peculiar œdematous look of the papilla also fades into a faintly-clouded appearance. The outline slowly becomes somewhat more sharply defined, but may remain obscure, passing insensibly into a faintly-clouded retina until the papilla has returned to nearly its normal level. The changes continue to progress; the disk becomes continually whiter, with more sharply defined outlines, until at last it is of a dead-white color, with hard margins which look as though cut by a punch, and with both arteries and veins atrophied. The retinal veins change much more slowly than do the arteries, and may remain dilated and tortuous even when the atrophy is considerably advanced.

Atrophy of the optic nerve may result from a lesion of the optic centres, or from pressure upon the optic nerve-trunk, chiasm, or tracts, as by a tumor, an injury, a local inflammation, or a distention of the third ventricle. Even meningitis may produce atrophy, in rare instances, without antecedent intraocular inflammation. These atrophies are white or gray. When gray, the tint may closely resemble that which is supposed to be more or less characteristic of sclerotic spinal disease. Primary atrophy not infrequently appears without known cause. Sometimes it is hereditary: in one instance it affected all the males of a family. Such cases have been studied by Leber, and more recently by W. F. Norris (*Trans. Amer. Ophthalmol. Soc.*, 1884).

The atrophy of the optic nerve which accompanies sclerotic conditions of the nerve-centres is not preceded by any stage of

swelling or of demonstrable inflammation. The normal grayish-pink tint of the disk begins to change into a peculiar bluish or bluish-green color, and at the same time the transparency of the disk diminishes, so that the retinal vessels are less readily traced into the substance of the papilla. As the atrophy progresses, the discoloration of the papilla gets more marked and its size may appear to be diminished; the outline grows continually harder and more sharply defined, and the choroidal border becomes excessively distinct, while within it the scleral ring grows unnaturally distinct and whiter than the adjacent nerve. The blood-vessels may diminish in size, the small ones upon the disk disappearing entirely. But such changes take place very slowly; and only in rare cases is there sensible lessening in the size of either the main arteries or veins. On the other hand, in other atrophies the arteries become very narrow, and at last are reduced to minute threads, and the veins slowly diminish in calibre. Greenish atrophy is developed so gradually, and the changes which it produces in the nerve are at first so slight, that its early recognition may be a matter of great doubt. To illustrate the difficulties of the situation, I may say that I once sent a patient to two of the best ophthalmologists of the country, and received absolutely antagonistic opinions.

Atrophy of the papilla from central nervous affections is accompanied by contraction of the field of vision, and the discussion of the diseases in which it occurs is deferred until the consideration of that symptom.

PUPILS.

In the examination of the pupils the first point to be attended to is their size. The norm of pupillary enlargement varies almost indefinitely in different individuals, so that it is impossible to detect slight departures from health. A pupil which is abnormally contracted is said to be *myotic*, the condition of contraction being known as *myosis*. The corresponding terms for enlargement of the pupils are *mydriatic* and *mydriasis*. From the adjectives myotic and mydriatic are formed the terms myotics and mydriatics, denoting classes of drugs which produce corresponding changes in the pupil.

Inequality of the pupils is of great diagnostic importance to

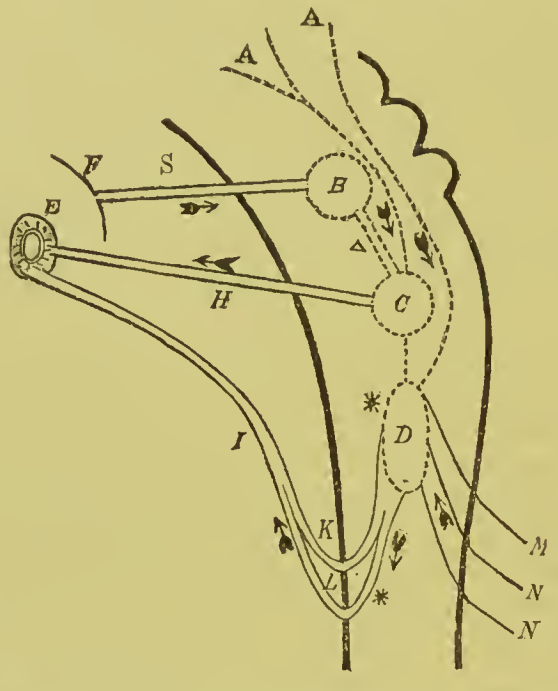
the neurologist, although it is occasionally present in normal eyes. I have also seen mistakes arise from one pupil being accidentally under the influence of a drug. The inequality of the pupils may be due to an excessive contraction or an excessive dilatation of one pupil. Which of these factors is the cause of the alteration is to be judged of by comparing the size of the pupils with that of the pupils of other persons. Excessive mobility of the pupil, a constant to-and-fro play without obvious cause, is an indication of nervous weakness, and especially of that kind of nervous mobility which is associated with hysteria. It is also seen in certain persons with a transparent, fine skin who are predisposed to tuberculosis.

The normal pupil contracts rapidly on exposure to light, and dilates with equal rapidity when the stimulus of the light is removed. When the eye is directed from a near to a distant object the normal pupil dilates, this dilatation being an associated movement with accommodation. Pinching the skin of the neck causes dilatation of the pupil, a phenomenon which is known as the *skin pupillary reflex.* A very peculiar series of pupillary phenomena is that which was first pointed out by Dr. Argyll Robertson as occurring in locomotor ataxia. In the *Argyll Robertson pupil* there is no movement when the skin of the neck is pinched, and no contraction or dilatation with the varying intensities of light, but the relations between the pupil and accommodation are preserved. In other words, a pupil which remains immovable when the skin of the neck is pinched, or when light is allowed suddenly to shine into the previously darkened eye, does move when the gaze of the patient is suddenly directed from a near to a distant object.

Loss of the light-reaction of the pupil when there is no poison in the blood nor evidence of cerebral congestion is often looked upon as proof of the existence of organic disease of the brain. As has, however, been shown by Thomsen (*Charité Annalen,* Berlin, 1885), after the epileptic paroxysm the pupil may be fixed for many hours, although consciousness has been completely recovered. Immobility of the pupil is also occasionally present in hysterical states, and, according to the researches of Uhthoff (quoted by Thomsen), it occurs in a small percentage of those who are insane but not paralyzed.

The explanation of the various pupillary phenomena is best made by means of the following diagram contrived by Erb.

Fig. 21.



A, nerve-fibres from the cerebrum. B, optic centre. S, optic nerve. E, pupil. F, retina. H, oculo-motor nerve. C, oculo-motor centre. D, ocular centres in the cervical spinal cord (cilio-spinal axis of Budge). IK and IL, sympathetic nerves. M, N, and N, sensory nerves.

Myosis.—Excessive contraction of the pupil, or myosis, may be due to paralysis of the roots of the sympathetic nerve in the cervical spinal region, or of the sympathetic nerve-fibres in the neck; or it may be the result of stimulation of the oculo-motor nerve-centres in the brain. In cervical spinal disease, such as pachymeningitis, myelitis, or chronic sclerosis, myosis may occur if the lesion is sufficient to paralyze. I have seen excessive unilateral myosis due to the pressure exerted upon the sympathetic nerve-fibres by enlarged cervical glands. Aneurisms and other tumors in the neck may have the same effect. If, in such a case, by the interference with circulation in the carotid, any cerebral symptoms are produced, or if there is hysteria, a mistake in diagnosis might readily be made.

A spasmodic myosis may be caused by an irritation of the oculo-motor centres or nerves. The contracted pupil of opium-poisoning and of cerebral congestion is due to centric irritation,

whilst that caused by Calabar bean is probably produced, at least in part, by a peripheral action.

Mydriasis.—Mydriasis, or dilatation of the pupil, may be due to irritation of the sympathetic, as in inflammatory lesions of the cervical spinal cord, but in the majority of cases it is the result of a centric or a peripheral paralysis of the oculo-motor nerve. The causes of such affections of the nerve have already been sufficiently discussed.

Action to Light.—The presence of the normal relations of the pupil to light proves in any case that the arc E C B F (Fig. 21) is intact,—*i.e.*, that the optic nerve and tract to the corpora quadrigemina and thence to the oculo-motor nucleus and thence through the oculo-motor nerve to the iris are functionally active. It must, however, be borne in mind that the two eyes act in association, so that contraction of both pupils occurs when light falls upon one eye. In this way a blind eye in which the optic nerve is paralyzed may have movements of the pupil. In examining an eye it is therefore essential that the other be covered.

Skin-Reflex.—It is believed that the skin pupillary reflex is produced through the spinal region. Thus, the impulse travelling up the cervical nerves stimulates the cervical spinal centres, which in turn send an impulse to the iris. This being the case, the preservation of the skin pupillary reflex shows that the arc M D I E is functionally active.

Movements of Accommodation.—Movements of the pupil with accommodation are brought about through the oculo-motor nerve, and are probably of the nature of habitual associated movements. They denote the integrity of the oculo-motor nerve and its nucleus.

Argyll Robertson Pupil.—In the Argyll Robertson pupil the failure of the pupil to contract under the stimulus of light shows that there is a lesion in the arc E C B F, or, in other words, that either the optic nerve or its centre, or the connection between the optic centre and the oculo-motor centre, or the oculo-motor centre or its nerve, is diseased. The retention of normal vision shows that the optic nerve and its nucleus are perfect. The occurrence of movements during the process of accommodation proves that the oculo-motor nerve and its centre are active: the interruption in the arc E C B F must therefore be between the optic and

the oculo-motor centre, or, in other words, in the commissural fibres which connect the optic and the oculo-motor centre. The loss of the skin pupillary reflex proves that there is some interruption in the arc M D L,—this interruption probably being in the spinal cord and due to the lesion which interrupts the continuity of the pathway between the oculo-motor and the optic centre.

Diseases in which the Pupils are deranged.—There are certain central nervous diseases of which the pupillary symptoms require more detailed discussion than has yet been given them. Prominent among these affections is *locomotor ataxia*, in which the pupils may be altered during the earliest stages. They may be either dilated or abnormally contracted. They may be equally affected, or one may be contracted and the other dilated, or one may be dilated and the other normal. According to Eulenburg, the simultaneous occurrence of myosis and mydriasis happens only in the late stages of the disorder. Myosis is much more frequent than mydriasis: thus, Eulenburg in sixty-four cases noted mydriasis in nine, myosis in twenty-eight. In the rare cases in which the cervical spinal cord is first attacked, myosis occurs very early. The degree of contraction varies greatly in different cases, but is often excessive: it is usually greater on one side, and often varies from time to time in the same case. T. Grainger Stewart states that he has seen the myosis increase during a gastric crisis, whilst Charcot affirms that during the pain of a crisis the pupil sometimes dilates.

In multiple cerebral spinal sclerosis the pupil is variously affected. With or without ptosis there may be dilatation of the pupil, or myosis may exist; in some cases there is inequality of the pupils. It is affirmed by Parinaud (*Progrès Médical*, vol. xii. p. 642) that there is a stage of the disorder in which the pupillary reflexes are exaggerated, and that if at the same time one eye is affected more than the other, monocular myosis may be produced whenever the patient goes into the light. According to the same author, whenever myosis exists in a person presenting symptoms of central nervous disease, if the pupil still reacts to light the cause is probably multiple sclerosis, and not locomotor ataxia.

The Argyll Robertson pupil is probably pathognomonic of degeneration of the upper spinal cord, including in this term the

medulla. It has been especially noted in locomotor ataxia, of which disease it is very characteristic. It also occurs in progressive paralysis of the insane. As, however, descending spinal degenerations are very common in general paralysis, the Argyll Robertson pupil is probably produced by these secondary spinal lesions, and not by the affection of the brain-cortex.

In general paralysis of the insane, the pupils may be equal and normal; or equal and contracted; or equal and dilated; or unequal on account of one being contracted and the other dilated, or on account of one being normal and the other dilated or contracted. Of all these phenomena, inequality of the pupils is most frequent and most characteristic. It may vary from day to day. One day the pupils may be equal, the next day they may be unequal; or to-day the pupils may be unequal because the right is contracted, and to-morrow they may be unequal because the right is dilated. This shifting inequality of the pupils is especially characteristic of the disease, and may be a prodromic symptom.

Dr. E. Mendel (*Paralyse der Irren*, Berlin, 1880, p. 147) states that he has seen inequality of the pupils as long as three and a half years before the outbreak of mental disturbance, whilst Foerster (quoted by Norris) relates the case of a colleague who, while yet of sound mind, jokingly said that on account of his pupils having become unequal he thought of taking quarters in an insane asylum, and who actually died a few years later in such an institution. Along with the dilatation of the pupil the shape of the eyeball may be affected. Mobèche and Mendel noted an increased convexity of the ball and a narrowing of the opening of the eye. The whole eye may also appear to be smaller than normal, on account of the paralysis of Mueller's muscles or of spasm of the orbicular muscle. In some cases ptosis or strabismus and double vision occur. After the epileptic attack of advanced progressive paralysis conjugated deviation of the eyes (Prevost's symptom, so called) is occasionally present.

DISTURBANCES OF VISION.

Method of Testing Vision.

In order to determine the acuteness of sight, test-types are employed, in which the letters are of various sizes, and numbered

according to the distance at which each size subtends a visual angle of five minutes, and the strokes of the letters an angle of one minute. This is considered to be a standard for average normal vision. The types constructed by Snellen, made upon this plan, are in common use, although many other series of test-types, equally useful, especially those in which the necessary alterations have been made to render them conformable to the metric system of measurement, are employed. When it is desired to test the acuity of vision, the patient should be placed twenty feet from the type-card, in a well-lighted room, and each eye tried separately. If the letters of No. XX are read, vision is normal, or 1, but if, standing at the same distance, no smaller letters than those numbered XL can be discerned, vision is $\frac{1}{2}$. It is usual to express these results according to the formula $V = \frac{d}{D}$, in which V stands for visual acuteness, d for the distance of the patient from the card, and D for the number of the type: so that in these instances the vision would be recorded $\frac{20}{XX}$ and $\frac{20}{XL}$. Twenty feet has been found to be a useful distance: any other may be chosen, provided it does not place the patient closer to the test-card than ten or twelve feet.

It often becomes a matter of importance to test the field of vision, or that space throughout which the eye is able to see while it remains stationary at a given distance from a fixed point. This may be roughly done by following the appended directions. Place the patient with his back to the source of light, and have him fix the eye under observation, the other being covered, upon the centre of your face, at a distance of two feet. Then move your fingers in various directions midway between yourself and him on a plane with your own face until you determine the limits of his indirect vision, controlling at the same time the extent and direction of your movements by your own field of vision. This plan of examination may be improved by placing small square pieces of white or colored paper on the end of a rod, and proceeding with the examination as before.

Although the field forms part of a hemisphere, it may be projected upon a flat surface and a useful map of the visual field obtained. Thus, let the patient be placed twenty-five centimetres

from a blackboard, which may be conveniently ruled in squares, and fix the eye under observation upon a small white mark. Then move the test-object, either a piece of white chalk* fastened in a black handle, or, better, pieces of white and colored paper one centimetre square, from the periphery towards fixation, until the object is seen or the color named; then mark this position. If eight peripheral points be marked and afterwards joined by a line, a fair map of the field of vision will be obtained, and may be transcribed upon a chart ruled for the purpose. This method is not entirely accurate if the field is larger than 45°, because beyond that angle, on flat surfaces, the object is too far away from the eye to make the examination exact.

FIG. 22.

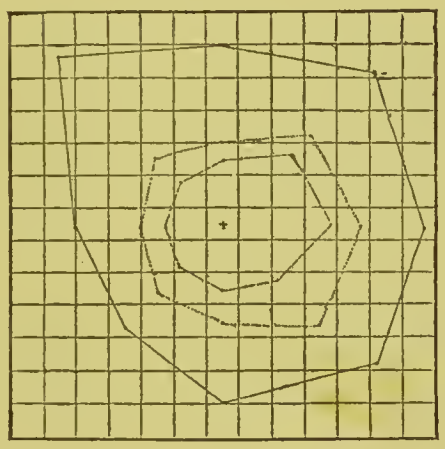


FIG. 23.



Fig. 22 represents the normal field of vision of the right eye taken on a flat surface: the outer marking is the boundary for white, and the others respectively for blue and red. For accurate measurements of the field, and for any measurements beyond 45°, an instrument known as a perimeter must be employed. This consists essentially of an arc marked in degrees, which rotates around a central pivot that is at the same time the fixing-point for the patient's eye. The test-object, square pieces of white and colored paper affixed upon a piece of dead-black cardboard, is moved from without inward, and the point in each meridian, where it is recognized, noted. The result is transcribed upon a chart prepared

* In amblyopic eyes the field may be taken in a dark room and a candle be substituted for the piece of chalk. In this way, so long as the patient can see at all, the field may be mapped.

by having ruled upon it radial lines to correspond to the various positions of the arc, and concentric circles to denote the degrees.

Fig. 23 represents the field as taken with the perimeter. The fields for color are smaller than the field for white, and are green, red, yellow, and blue from within outward. Landolt's investigations—those usually quoted—make the normal fields for color as follows:

	Blue.	Red.	Green.
Upper	50°	35°	30°
Outer	80°	70°	55°
Lower	55°	45°	35°
Inner	50°	40°	30°

As many meridians may be tested as are needful: four are usually sufficient.

Disorders of Sight.

The disorders of vision which require study by the neurologist are—amaurosis, or nerve-blindness, including amblyopia, or impaired vision; hemianopsia, or loss of vision over one-half (lateral or vertical) of the eye; scotomata, or patches of blindness; and contraction of the field.

Anatomy of Optic Tract.—Before discussing these various visual disturbances it is perhaps well to summarize briefly the anatomy of the optic tract. The band of white fibres known as the optic tract arises from the posterior part of the thalamus, the geniculate bodies, and the superior quadrigeminal bodies. From the under part of the thalamus it suddenly bends forward, and as a flattened band passes obliquely inward across the upper anterior surface of the cerebral peduncle, to which it is closely attached; after this it adheres to the tuber cinereum, from which and from the lamina cinerea it is said to receive fibres. In this way it reaches the optic chiasm, an oblong flattened body situated upon the olivary eminence of the sphenoid bone. In the chiasm the fibres of the nerves coming from the tract divide: the larger or outer band decussates with its fellow, or, in other words, crosses over to enter the optic nerve as it emerges from the opposite side of the chiasm and to be finally distributed to the nasal half of the retina. The inner or smaller band of fibres passes on through to the chiasm, without decussation, to the outer side of the retina of its own eye. These fibres are the only ones that have direct relations with vision,—

the *inferior commissure* of Gudden (the posterior loop of Hannover) being composed of fibres which pass through the optic tracts and the posterior portion of the chiasm to connect the two inner geniculate bodies, whilst the existence of the inter-retinal fascicule, which Hannover believed to pass through the anterior edge of the chiasm and to be a retinal commissure, is denied by most recent anatomists. Although the optic tracts are closely connected with the corpora geniculata and the anterior corpora quadrigemina, it seems clearly made out that these centres have not the function of vision. The conscious perception of retinal images takes place in the brain-cortex. The exact position at which it occurs has been much discussed, but the consideration of this is so closely bound up with the question of hemianopsia that it is better postponed for the present.

Amaurosis.—Amaurosis was the term employed by the old surgeons to designate cases in which they could find no cause in the eye for the loss of sight. In recent times it has come to signify blindness from disease of the nerve or of the nerve-centres. Amblyopia is a partial loss of vision of nervous origin. The two terms are sometimes rendered synonymous by means of adjectives: thus, partial amaurosis is used to signify amblyopia, and complete amblyopia is sometimes employed instead of amaurosis. There is at present sufficient clinical evidence to show that a temporary amaurosis may be produced by a distant irritation. The researches of Dr. Brown-Séquard (*London Lancet*, July, 1861) proved, many years ago, that amaurosis may occur in diseases of the cerebellum without alteration of the nerve. Dr. Davaine is said to have reported twelve cases of amaurosis produced by intestinal worms. Dr. Brown-Séquard has seen it in animals following injury to the spinal cord, and has noticed its occurrence in man as the result of irritation of the nerves of the stomach.

Amaurosis may also be an hysterical symptom. Under these circumstances, its true nature is usually revealed by the suddenness with which it develops and disappears, as well as by the coexistence of other pronounced hysterical symptoms. (See, also, page 259.)

Organic Amblyopia.—Partial or complete neurotic blindness may be due to lesions of the peripheral visual nervous system or to centric diseases. For reasons that will become manifest during

the study of hemianopsia, a lesion of the brain which directly causes loss of vision by acting on the visual centres almost invariably affects only a portion of the field in both eyes, causing hemianopsia. A central lesion which produces general amblyopia, affecting the whole field of each eye, must be double and implicate both cerebral hemispheres. Such a lesion is so excessively rare that it may be laid down as a diagnostic rule that an organic amblyopia is due to peripheral disease. Such disease may be a gummatous, gliomatous, or sarcomatous tumor so situated as to press upon the optic chiasm or the optic tract at the base of the brain. It may be an inflammation of the optic nerve, either a true neuritis or a choked disk. Almost invariably inflammations of the optic nerve are symmetrical and affect alike both eyes.

Toxæmic Amblyopia.—Toxæmic amblyopia is usually of organic origin, but, as the lesion is directly produced by the poisoning, and is likely to be recovered from on removal of the poison from the system, the separation of toxæmic amblyopias is of practical importance. The most common and the most important of the class is the loss of vision produced by *tobacco.* In a large proportion of cases the excess in the use of tobacco has been associated with an excess in the use of alcohol, and there has been much discussion as to which of these agents was the cause of the optic derangement. The amblyopia is frequently present in those who smoke excessively but do not drink, and tobacco seems to exert the more potent influence. The victim of tobacco amblyopia shows no difficulty or awkwardness in going about, but especially complains that vision is very bad in direct sunlight. He almost invariably sees better on dull days, and in the early morning and evening. If this be not noted by the patient himself, it may be shown by testing vision with type in full daylight and again in a darkened room. An examination of the visual field will show that a great functional defect is in the centre of the field, occupying an oblong or oval patch which extends from the fixing-point (corresponding to the macula lutea) out towards and often immediately beyond the blind spot (corresponding to the disk). This central scotoma is relative, and not absolute; *i.e.*, loss of vision in it is never complete. It is especially marked for the perception of colors, for green and red in particular: the former is usually described by the patient as "white" or "gray," and the latter as

"brown" or "no color at all." In most cases the scotoma is smaller than the visual field for central colors, green and red, and hence a zone is present beyond the scotoma in which these colors are observed. This is especially the reason that the patient will recognize the color of a large body and mistake that of a very small one.

The scotoma of tobacco amblyopia is invariably anatomically symmetrical, occupying exactly the same position in each retina. It is believed by oculists to be chiefly due to the change in the peripheral portion of the nerve-axis. It has been asserted in the rare cases in which the scotoma is central and surrounds the fixation-spot equally on all sides that the cause is alcohol. Dr. Edward Nettleship (*St. Thomas's Hospital Reports*, vol. ix.) states, however, that in all the cases of such scotoma which he has seen the patients were smokers. And Dr. G. De Schweinitz has reported (*Philadelphia Medical Times*, 1886) an example of such scotoma in a woman who used neither alcohol nor tobacco, but made cigars, and in whom the eyes became normal after she left her occupation. In investigating a case it should be remembered that chewing tobacco is more injurious than smoking.

Cases of blindness directly produced by *lead* are stated to occur. Such cases, however, must be very infrequent, since I have seen a great many cases of lead-poisoning, but never such a one. More frequent is the indirect production of blindness by lead. Thus, the saturnine Bright's disease may cause a uræmic degeneration of the retina, and in cases of violent saturnine brain-disease (*encephalopathia saturnina*) choked disk is usually present, with consequent loss of vision. According to Norris (*System of Practical Medicine*, Philadelphia, 1886, vol. iv. p. 804), "excessive overdoses of *quinine* impair the sight, and in some cases have produced temporary but absolute blindness. The usual symptoms are a deterioration of the central vision, with contraction of the field. The ophthalmoscopic examination reveals a pallid disk, with marked diminution in the size of the retinal arteries and veins." Many years ago I saw a complete blindness, lasting eight or ten hours, produced in a young lady by fifteen grains of sulphate of quinine: that the alkaloid was the cause of the symptoms was proved by their recurrence on a repetition of the dose. *Santonin* in toxic doses pro-

duces dilatation of the pupil and great disturbance of vision. (See author's treatise on *Therapeutics;* also *Virchow's Archiv,* Bd. xx., 1860; Bd. xxviii., 1863.) The action of *salicylate of sodium* so closely resembles that of quinine that it is probable amblyopia might be caused by it; and Gatti (quoted by Norris) reports a case of transient amblyopia attributed to the ingestion of one hundred and twenty grains of salicylate of sodium.

Hemianopsia.—Hemianopsia is a loss of vision in one-half of the eye. Since the rays of light cross in the eye, the part of the retina which is blind is always opposite to the object which cannot be seen. Thus, when the eye sees no objects to the left of it, the symptom is termed left hemianopsia, although the blind spot is on the right side of the retina: in other words, when an object in front of the line of the nose is not seen, the hemianopsia is spoken of as nasal, although the temporal half of the retina is paralyzed.

Hemianopsias are best divided for our purposes into horizontal, in which the dividing line between the paralyzed and the active portion of the retina is horizontal, and vertical, in which the dividing line is vertical.*

Horizontal hemianopsia may be inferior or superior. It is almost always due to disease of the eye. The only known nerve-lesion capable of producing it is a tumor or other alteration of the hemisphere so situated and developed as to press downward upon one optic tract. Such cases have been recorded.

Vertical hemianopsia is almost invariably due to nerve-lesion. A large number of terms have been employed to designate the varieties of vertical hemianopsia. Of these the following seem worthy of adoption:

a. Temporal hemianopsia, in which both temporal fields are involved.

b. Nasal hemianopsia, in which both nasal fields are involved.

c. Lateral or *homonymous hemianopsia,* in which corresponding

* The term *hemiopia* has been used by some writers as synonymous with hemianopsia; but Dr. Seguin defines it, "as now accepted," to signify loss of perceptive power in one-half of the retina, whilst hemianopsia means obscuration of one-half of the visual field. A right hemiopia is equivalent, therefore, to a left hemianopsia. To avoid confusion, I shall not use the term hemiopia.

fields of the eyes are involved: thus, both left or both right fields are destroyed.

Fig. 24.

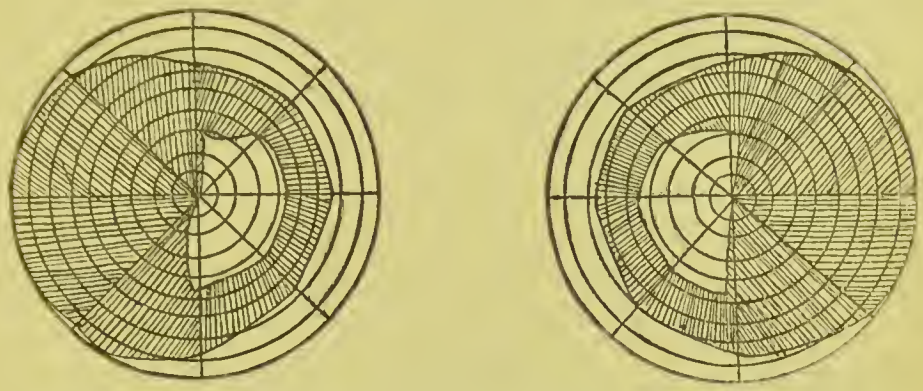


Diagram of the fields of vision in a case of temporal hemianopsia. The shading represents the areas of absent vision; the outer boundary of the shading is the limit of the normal field. The patient was aged 40, had brain-syphilis, and probably a deposit upon the chiasm. V = ½. Both optic nerves gray-green in color and atrophic.

Both temporal and nasal hemianopsia are exclusively caused, so far as our present knowledge goes, by lesion of the optic chiasm.

Fig. 25.

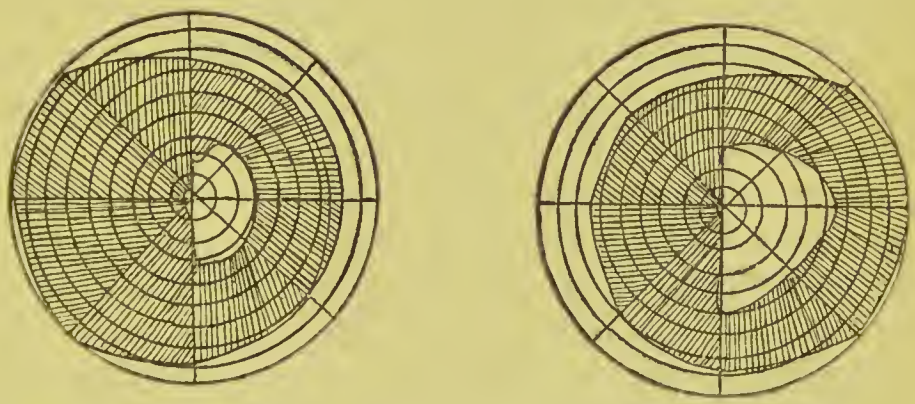


Diagram of the fields of vision in a case of left lateral hemianopsia. The outer boundary of the shading is the limit of the normal field; the shading represents where vision was lost. The left half of each field is absent, and the right halves are contracted. The patient was a woman, aged 45. V = ¼. Optic nerves oval, gray in their deeper layers, but superficially capillary. Central circulation unchanged. Seven months before examination the patient had a temporary left-sided hemiplegia.

Lateral hemianopsia appears to be always produced by lesions of one optic tract, or of the more central parts of the optic apparatus as far posterior as the cortical centre for vision in one hemisphere. Central lesions causing lateral hemianopsia are usually embolic or hemorrhagic, and hence are apt to be suddenly developed. Lesions of the optic tract causing lateral hemianopsia

are probably always slowly progressive, being of the nature of the growths or exudations.

A study of the diagram on the opposite page, after Prof. Seguin, will show the correctness of the following deductions:

1. When a lesion involves one optic tract it causes lateral hemianopsia, the side of the hemianopsia being opposite to the side of lesion. Thus, if the right tract is pressed upon, a left lateral hemianopsia results.

2. A lesion acting upon one side of an optic tract so as to compress only some of its fibres might produce one-sided hemianopsia, either nasal or temporal, according to the fibres involved. If the pressure be upon the outer side, the hemianopsia will be nasal; if it be on the under side, the hemianopsia will be temporal. Thus, tumor T 1, in the diagram, acting only feebly, would cause a unilateral nasal hemianopsia; T 2, a unilateral temporal hemianopsia; although either tumor, if sufficiently large to compress the whole tract, would cause an homonymous right hemianopsia.

3. When both sides of the optic chiasm are pressed upon by a lesion not sufficiently powerful to obliterate the function of the chiasm, both lateral fasciculi are pressed upon, and a double nasal hemianopsia results. Thus, the tumors T 3 and T 4 acting together would cause a double nasal hemianopsia.

4. When the lesion involves the frontal and posterior borders of the chiasm it injures the decussating nerve-fibres, and thereby causes double temporal hemianopsia. Thus, the tumors T 5 and T 6 acting together would cause a double temporal hemianopsia.

These forms of hemianopsia are almost invariably accompanied by changes in the optic disk and other evidence of pressure or change in the basal nerves, which symptoms are very apt to be wanting in lesions that involve the cortical centre.

The most elaborate study which has been made of lateral, cortical hemianopsia is that of Prof. E. C. Seguin (*Journal of Nervous and Mental Diseases*, vol. xiii.). In this paper sixteen cases are analyzed, leading, as Dr. Seguin believes, to a final determination of the position of the cortical centres which are connected with vision. These cases show—

"That lesions of the corpus geniculatum laterale, pulvinar, and latero-caudad, etc., of the thalamus may cause hemianopsia,—

usually in association with hemianæsthesia and hemiplegia, or with hemianæsthesia alone.

Fig. 26.

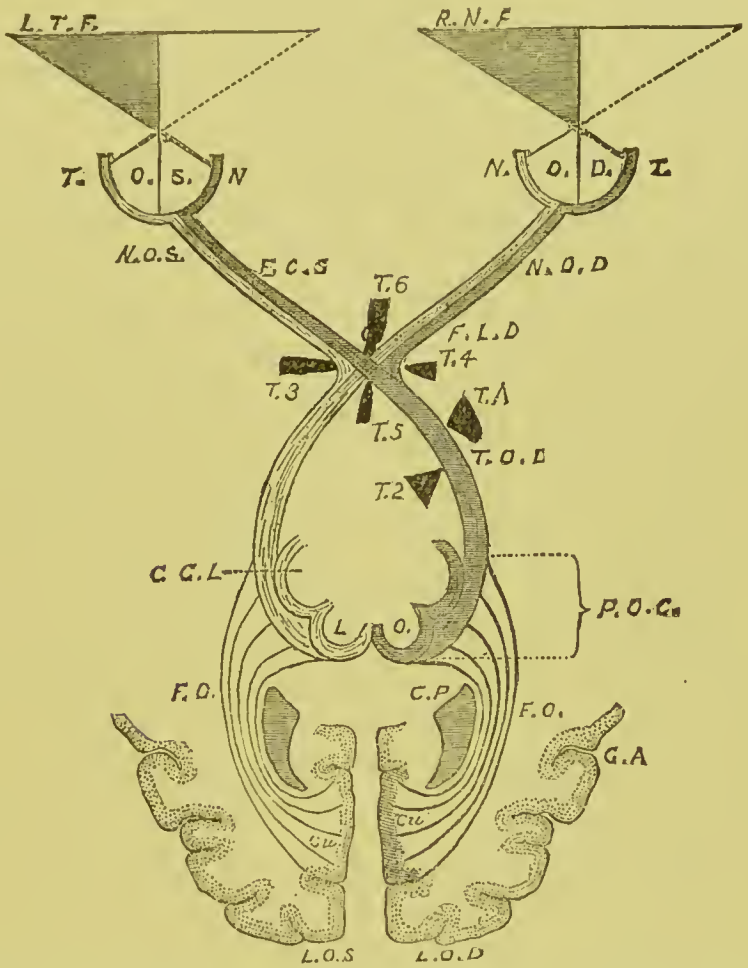


Diagram of Visual Paths; designed to illustrate specially Left Lateral Hemianopsia from any lesion. L. T. F., left temporal half-field. R. N. F., right nasal half-field. O. S., oculus sin. O. D., oculus dexter. N. T., nasal and temporal halves of retinæ. N. O. S., nervus opticus siu. N. O. D., nervus opticus dext. F. C. S., fasciculus cruciatus sin. F. L. D., fasciculus lateralis dext. C., chiasma, or decussation of fasciculi cruciati. T. O. D., tractus opticus dext. C. G. L., corpus geniculatum laterale. L. O., lobi optici (corpus quad.). P. O. C., primary optic centres, including lobus opticus, corp. genic. lat., and pulvinar of one side. F. O., fasciculus opticus (Gratiolet) in the internal capsule. C. P., cornu posterior. G. A., region of gyrus angularis. L. O. S., lobus occip. sin. L. O. D., lobus occip. dext. Cu., cuneus and subjacent gyri constituting the cortical visual centre in man. *The heavy or shaded lines represent parts connected with the right halves of both retinæ.*

"That a lesion of the white substance of the occipital lobe in the caudal radiations of the internal capsule may cause hemianopsia, alone or with hemianæsthesia.

"That lesions of the supra-marginal gyrus, angular gyrus, and inferior parietal lobule with the subjacent white substance may

cause hemianopsia, with or without other symptoms (hemiplegia, loss of muscular sense, word-deafness, etc.).

"That a lesion of greater extent, involving the speech-centre, the motor convolutions, and the parts enumerated above (4), due usually to embolism or thrombosis of the entire Sylvian artery, will, when existing on the left side, produce aphasia, alexia, hemianopsia, and hemiplegia.

"That lesions of the occipital lobe, cortex, and subjacent white matter produce blindness when bilateral, and hemianopsia when unilateral. This conclusion is in accord with Exner's (1881)."

It will be remembered that there are two distinct views held by physiologists, Munk teaching that the centres for conscious visual perception are in the occipital lobe, and Ferrier that they are in the angular gyrus. The elaborate discussion of the physiological evidences is out of the province of the present article, but the explanation of these apparently discordant results as given by Dr. E. C. Seguin is probably correct,—namely, that the white band of conducting fibres known as the optic fasciculus of Gratiolet and Wernicke, whilst passing from the posterior part of the thalamus to the cuneus of the occipital lobe, lies so close to the inferior parietal lobule and the angular gyrus that a lesion of the angular gyrus or of the supra-marginal gyrus, or even of the inferior parietal lobule, might press upon or otherwise involve this conducting fasciculus, and thus interrupt the communication between the perceptive visual centres and the eyes. Both Ferrier and Munk may, therefore, be correct in their views,—the loss of sight in Ferrier's experiments being due not to the wounding of the angular gyrus itself, but to the interference with the band conducting the white matter beneath the gyrus.

The following rules for the diagnosis of the seat of the lesion in cases of hemianopsia are those of Dr. Seguin, and seem to me correct:

"1. Lateral hemianopsia always indicates an intra-cranial lesion on the opposite side from the dark fields.

"2. Lateral hemianopsia, with pupillary immobility, optic neuritis, or atrophy, especially if joined with symptoms of basal disease, is due to lesion of one optic tract, or of the primary optic centres on one side.

"This diagnosis may be further strengthened and rendered quite

certain by seeking for and finding one-sided pupillary reaction, as recently suggested by Wernicke. He ingeniously predicts that only one lateral half of each iris will be found to contract by the reflex effect of light when one optic tract has been interrupted. He designates this as 'hemiopic pupillary reaction.'

"3. Lateral hemianopsia, or sector-like defects of the same geometric order, with hemianæsthesia and choreiform or ataxic movement of one-half of the body without marked hemiplegia, is probably due to lesion of the caudo-lateral [posterior lateral] part of the thalamus, or of the caudal division of the internal capsule.

"4. Lateral hemianopsia, with complete hemiplegia (spastic after a few weeks) and hemianæsthesia, is probably caused by an extensive lesion of the internal capsule in its knee and caudal [posterior] part.

"5. Lateral hemianopsia, with typical hemiplegia (spastic after a few weeks), aphasia if the right side be paralyzed, and with little or no anæsthesia, is quite certainly due to an extensive superficial lesion in the area supplied by the middle cerebral artery: we would expect to find (as in Case 26 by Westphal) softening of the motor zone and of the gyri lying at the extremity of the fissure of Sylvius,—viz., the inferior parietal lobule, the supramarginal gyrus, and the gyrus angularis. Embolism or thrombosis of the Sylvian artery would be the most likely pathological cause of the softening.

"6. Lateral hemianopsia, with moderate loss of power in one-half of the body, especially if associated with impairment of muscular sense, would probably be due to a lesion of the inferior parietal lobule and gyrus angularis, with their subjacent white substance penetrating deeply enough to the visual centre.

"7. Lateral hemianopsia, without motor or common sensory symptoms, this symptom alone, is due, I believe, from the convincing evidence afforded by Cases 28, 29, 41, and 45, to lesions of the cuneus only, or of it and the gray matter immediately surrounding it on the mesal surface of the occipital lobe in the hemisphere opposite to the dark half-fields. Most surgical cases come at once, or after convalescence, within this rule, or in No. 6 (Case 3)."

Contraction of the Field of Vision.—Contraction of the

field of vision occurs in several forms of selerosis of the nervous system, and is probably the result of secondary organic changes in the optie nerve. It is especially frequent in locomotor ataxia, but may occur in multiple sclerosis. The contraetion is concentric, and, according to Forster, in a majority of cases is generally more marked on the outer side. The curve that bounds the field of vision is usually irregular with emarginations, which have a tendency to take the form of sectors whose centre is the optic papilla. The contraetion of the field progresses steadily and with greater rapidity than the loss of sight, so that vision may be satisfactory although the sensitive portion of the retina is almost limited to the maeula. Microscopic studies of the optic nerve have shown that the eontraction of the field is connected with a degeneration of the nerve-fibres which commences in the outer portions of the optic nerve and travels towards its eentre. Disorder of the color-sense usually accompanies the eontraction of the field. The power of pereeiving yellow and blue is preserved for a long time, whilst blindness for green and red is early developed. Aecording to M. Abadie, it is possible to distinguish between parenchymatous and interstitial atrophy of the optic

Fig. 27.

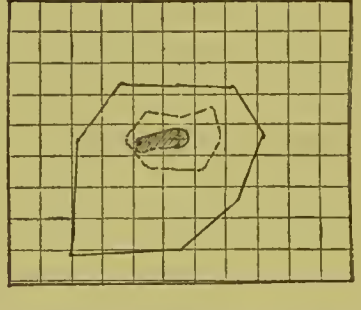


Diagram of the fields of vision for white and red and central scotomata, embracing fixation and the blind spot, from a case of locomotor ataxia. Patient aged 58; V=$\frac{1}{10}$. Both optic nerves showed gray atrophy. Inasmuch as he smoked two ounces of tobacco a week, and drank "a few" glasses of whiskey daily, the influence of these in producing the scotomata cannot be entirely excluded. For a similar case see Gowers, *Medical Ophthalmoscopy*, p. 111, Fig. 20.

nerve by a study of the relation between the general sight and the eolor-sense. When the loss of vision is still greater than $\frac{1}{5}$, if green is no longer pereeived, and red and yellow are recognized with difficulty, there is parenchymatous atrophy; if, on the other hand, with an acuteness of vision inferior to $\frac{1}{10}$, the

perception of color is satisfactorily maintained, there is probably interstitial atrophy.

Multiple sclerosis may produce disturbances of vision similar to those caused by locomotor ataxia. Out of fifty cases observed by Dr. R. Gnauck (*Neurolog. Centralblatt*, 1884, iii. 315), vision was affected in twenty-two. In some cases the disturbance of vision is monocular, probably as the result of the development of a focus of sclerosis in the nerve itself. According to Parinaud (*Progrès Médical*, xii. 642), the amblyopia of multiple sclerosis, unlike that of posterior sclerosis, very rarely ends in total blindness; but in two of Gnauck's cases the loss of sight was complete.

SENSE OF TASTE.

The function of taste is shared by two nerves,—the glosso-pharyngeal and the lingual branch of the fifth,—the first supplying the posterior half or two-thirds of the tongue, the last the anterior half or third of the organ. Owing to this double nerve-supply, it is necessary, in testing the condition of the taste-sense, that the tongue be protruded from the mouth and be kept quiet after contact with the sapid substance until time has elapsed for the penetration of the latter. The gustatory filaments of the lingual branch of the fifth leave the nerve with the chorda tympani and pass to the facial nerve. Paralysis of the lingual branch of the fifth nerve after it receives the chorda tympani is therefore followed by loss of taste in the anterior part of the tongue; as is also a lesion of the trunk of the facial nerve between the geniculate ganglion and the point at which the chorda tympani separates from the facial nerve. There have been cases of paralysis of the facial nerve above the geniculate ganglion in which there has been no interference with the sense of taste, and, on the other hand, cases have been reported in which compression of the trigeminus nerve above the position at which the petrosal nerves join it has been followed by loss of taste. It is therefore probable that the gustatory fibres of the lingual nerve return through the petrosal nerves to the trunk of the trigeminus. There are, however, cases on record in which the taste-symptoms are very difficult of explanation, and it is possible that the course of the filaments varies.

The glosso-pharyngeal nerve arises from the nucleus in the

medulla close to the nucleus of the vagus. The trunk emerges in the groove between the olivary body, and escapes from the skull through the jugular foramen.

Hyperæsthesia of the sense of taste is sometimes seen in hysteria. Hallucinations occur in insanity, and with extreme rarity are produced by organic disease of the nervous apparatus involved. They are very frequently present as the result of disorders of the intestinal tract: whether under these circumstances they should be looked upon as reflex or as the development of abnormal mouth-secretions is doubtful.

SENSE OF SMELL.

It is necessary in testing the sense of smell that such odorous substances be selected as are not irritant to the mucous membrane of the nose, lest the subject should detect their presence by their effect upon the branches of the trigeminal nerve in the nasal mucous membrane. Hyperæsthesia of the sense of smell is sometimes seen in hysteria, and theoretically should occur in inflammatory conditions of the olfactory tract, but I have never known of such a case. Hallucinations of the sense are sometimes seen in insanity, and also occur in diseases of the olfactory tract, when they are apt to usher in an epileptic convulsion. I have seen such symptoms produced by a gliomatous tumor involving the olfactory lobes. Loss of smell usually depends upon disease of the mucous membrane of the nose, but it may be caused by an affection of the olfactory lobes, and is occasionally seen in organic hemianæsthesia produced by a lesion involving the internal capsule.

CHAPTER IX.

DISORDERS OF MEMORY AND CONSCIOUSNESS.

ALL functional acts are accompanied by, or dependent upon, a nutritive disturbance. It matters not whether the functional act is connected with thought, consciousness, or secretion, the generation of nerve-force by the ganglionic cell and its transmission by nerve-fibre are accompanied by nutritive changes in these bodies. A nutritive act, although temporary, has a distinct tendency to impress permanently the part implicated; and this tendency is especially pronounced in nervous tissue. All nervous tissue is, therefore, liable to be permanently affected by its own functional actions. This, it must be remembered, applies equally to normal and to pathological activities. Thus, the child in learning to walk by repeated efforts trains the lower nerve-centres until, in response to appropriate stimuli, a definite series of nervous discharges and transmissions occur independently of the will, and walking becomes automatic. This, in short, is the history of all training, mental and physical. All nervous tissues, therefore, have memory, —*i.e.*, the faculty of being permanently impressed by temporarily acting stimuli, the thing remembered being, in fact, the functional excitement.

The recognition of the universality of memory in nerve-tissues is of great importance in the consideration and treatment of disease. Thus, an epileptic fit is produced by a peripheral irritation. If that peripheral irritation be at once removed, the fit does not recur, and the patient is cured. If, however, the irritation be not soon taken away, but produce a series of convulsions, the fits may continue after the removal of the irritation, simply because of the permanent impress which has been made upon those cells in the brain-cortex whose discharge of nerve-force is the immediate cause of the epileptic paroxysm. The nutrition of the cells has been so altered that at irregular intervals they fill up and discharge nerve-force.

Owing to this power of memory, a physical habit may become

so permanently engrafted upon the nervous system that the patient is unable to control it. An example of this is seen in the so-called habit-choreas: movements at first controllable, mere bad habits, become at last fixed, not to be altered by any power. The hysterical woman who gives way to hysterical nervous impulses thereby strengthens their hold upon the system, so that in time she may lose all power of control over the lower nerve-centres. Moral habits are formed in obedience to the same law. Self-control, enforced at first by discipline, may become at last in the child an integral function of the nervous centre, by a method parallel to that by which an accidental epilepsy is converted into a permanent disease. In the prognosis and treatment of disease, as well as in the training of the young, the full recognition of the power of habit—*i.e.*, of unconscious memory—is a matter of vital importance.

What is true of the lower nerve-centres and fibres is true of the upper ones. Intellectual acts or thoughts and perceptions tend to stamp themselves upon the centres connected with them, and when the function of the nerve-cell is connected with consciousness the changes which occur in the nutrition give origin to conscious memory,—*i.e.*, to memory in the usual sense of the term.

The methods of ordinary mental action seem to indicate either that special ganglionic cells are set apart for special forms of memory, or else that the single ganglionic cell is capable of distinct acts of memory. Thus, one individual will remember one class of facts with great ease, to the exclusion of other matters, whilst the second person may readily remember those affairs which the first naturally forgets. Disease sometimes dissects out, as it were, the different forms of memory, isolating one from the other. It is well known that in the loss of memory which accompanies senile changes of the brain, or is a prominent symptom in the first stage of general paresis, the power of remembering recent events may be lost, although the recollection of affairs which happened in childhood days is far more vivid than in the normal condition of the individual. Under these circumstances it may be considered that the ganglionic cells have lost their capability of receiving impressions, but not of recognizing impressions which were made long before. The separation of different

forms of memory is, however, distinct from this. Thus, in a case of dementia recently under my care memory for ordinary events was almost entirely lost, and yet a joke or a ludicrous story would be remembered in all its details without apparent effort. It is well established that one form of memory—namely, that connected with language—has in most individuals a definite brain-location; and it may be that each variety of memory has its own territory.

In considering the disorders of memory I shall discuss, first, disturbances of specialized forms of memory; secondly, disturbances of the general function. This arrangement may appear to be a reversal of the natural order, but the peculiar relations of memory to consciousness, and the directness with which a discussion of either of these functions leads to a consideration of the other functions, render the plan which I have selected the more convenient. Of the special forms of memory which presumably exist, the only one whose symptomatology is sufficiently worked out to necessitate discussion here is that connected with language.

WORD-MEMORY.

The power of speech may be lost by an individual from paralytic or other affections of the larynx preventing the formation of sound; from paralysis of the tongue and lips causing inability to pronounce words or letters; and, finally, from derangement of the mental functions immediately connected with word-thought. To the voicelessness of laryngeal disease the name of *Aphonia* may be applied; to that which is the result of affections or paralysis of the tongue or mouth, *Aphæmia*; whilst for the mental difficulty may be reserved the term *Aphasia*. These terms have, however, been employed with various significations by various authors. Aphæmia has been used to signify aphasia, and the condition which I have called aphæmia is known by some writers as ataxic aphasia,—a term which is, however, used by other writers to denominate a peculiar variety of aphasia. With aphonia and aphæmia we have at present no concern.

Aphasia.—In aphasia the power of verbal expression or of word-perception is affected, although the general intelligence of the patient may be intact. In the completest form of aphasia,

when the patient can neither understand spoken or written language, nor express himself either in words or in writing, the intelligence is to be judged of by the acts of the patient: thus, such an aphasic will understand the use of the pen, and perhaps attempt, if he be not paralyzed, to write, or he will use properly a spoon or a knife, although he has no knowledge of the language or written symbols habitually employed to represent such articles.

Aphasia is in many cases not complete,—that is, it does not affect all the mental functions connected with speech. In 1880, Dr. Magnan divided the cases of aphasia into two groups: first, those cases in which all connection between words and their meanings is lost, so that the aphasia is complete; to these cases he gave the name of *Verbal Amnesia:* secondly, cases in which the subject is able to comprehend spoken language, but has lost the power of expressing himself; thus, when asked to pick up a pen from the table, the patient does so, but holding the pen is unable to name it: to this partial or incomplete aphasia Magnan gave the name of *Logoplegia.*

As long ago, however, as 1843, Dr. Lordat, of Montpellier, reported cases of aphasia, and clearly recognized the existence of much more partial and distinct forms of the affection than those outlined by Magnan; and in 1874, Wernicke, under the name of *Sensory Aphasia*, described certain peculiar cases similar to those noted by Lordat. In 1874, Kussmaul also reported cases of sensory aphasia. Magnan and Kussmaul have disputed much concerning their rights to priority, but they each were long antedated by Dr. Lordat.

Kussmaul (*Ziemssen's Cyclopædia*, vol. xiv.), in an extraordinarily elaborate and difficult-to-be-read article upon speech, divides aphasia as follows:

First. Ataxic Aphasia, or that condition in which the patient has an entire loss of speech, although written and spoken language is understood. In pure ataxic aphasia there is no *agraphia*, or loss of the power of writing, so that the subject may be able to transact by writing the most complex business. When agraphia is added to ataxic aphasia the patient is entirely unable to communicate with his fellows except by rude signs. Agraphia is not dependent upon any loss of control over the finer move-

ments of the hand. Thus, in a case reported by Spamer, a young woman completely agraphic was still a skilful seamstress. The facial expressions of emotion are usually preserved, so that anger, sorrow, etc., are expressed in the face, and sign-language—indeed, the whole mimetic faculty—may be normal. In very rare cases sign-speech is also lost, so that the patient is no longer able to communicate by pantomime.

Second. Amnesic Aphasia, in which the idea is present but the word is wanting, although articulation is "at the service of the word." Under these circumstances the patient is unable to talk, although he can still repeat and also write out words which are spoken to him. In partial amnesic aphasia proper names are the first to be lost, next names of things, and finally nouns in general. The patient will often paraphrase the noun which he cannot remember. Thus, a pair of scissors may be called "that with which one cuts;" the window, "that through which one sees." The early forgetfulness of proper names is evidently founded upon their isolated arbitrary character, which fails to link them with the world of word-thought, and renders them even in health apt to be forgotten. In some cases of amnesic aphasia it is only certain letters that are left out. Thus, L. Schlessinger details the case of a boy who invariably omitted the initial consonants, both in writing and in speaking.

Third. Word-deafness and *word-blindness*, constituting the *Sensory Aphasia* of some authors. The subject of *word-blindness* is able to express his ideas in conversation with his normal fluency, and to understand all that is said to him, also to copy written language and with the pen to put down upon paper his ideas, yet he is unable to read printing or writing. Thus, in a case reported by Charcot the patient suffering with partial right hemiplegia was unaware that he had any disorder of word-thought until he wished to give certain orders about his business affairs. These he wrote, and afterwards, in order to be sure of their correctness, undertook to read what he had written, when he found that he could not recognize a single word.

In *word-deafness*, although the sense of hearing is intact, and although the individual is able not only to express himself in conversation, but also to write, to read, and to understand writing, he comprehends nothing that is said to him by word of mouth.

He hears distinctly, but cannot connect the sound of the word with the object which it symbolizes.

Fourth. Paraphasia, or that condition in which the person is unable to use words in their proper sense, speaking, it may be, with considerable fluency, but perpetually using one word for another.

Fifth. Agrammatisma or *Akataphasia,* in which all the rules of grammar are lost, so that the parts of speech are hopelessly intermingled.

It seems logical to consider the fourth and fifth varieties of aphasia of Kussmaul as simply partial aphasias, in which the relations of words with ideas have become dislocated but not completely disjoined. If all peculiar forms of partial aphasia must be defined and named, the list will have to be much extended beyond that of Kussmaul. Thus, I might instance a case reported by Dr. Grashey (*Sitzungsberichte Med. Gesell. Würzburg,* 1884, No. 9), in which the patient understood conversation, and read and wrote freely, but in attempting to talk was frequently unable to remember words, except by writing and then reading them. It was his common practice to write with his forefinger upon the palm of his left hand each word, letter by letter, and then immediately pronounce it. I have heard or read of a case in which the patient repeated backward the sentence that he was trying to utter, beginning at the end of the sentence and at the end of each word. Instead of "John is a bad boy," the patient would say, "Yob dab a si nhoj."

As was pointed out by Dr. Hughlings-Jackson in 1864 (*Lond. Hosp. Report,* 1864), the faculty of intellectual speech may be lost whilst emotional speech remains to a greater or less extent. Thus, a person who has been considered completely aphasic, after obstinate silence to all questions and remarks, will, in a burst of anger, suddenly swear violently. Or, as in a case seen by Dr. Jackson, an aphasic who is unable to respond "no" to an ordinary question blurts out the monosyllable when the question is so worded as to provoke his anger. Dr. Broadbent reports a case in which the power of voluntary speech was entirely lost, but in which under emotion a large number of words would be forced out rapidly like so many interjections. Brown-Séquard states that aphasic persons sometimes recover their speech during de-

lirium, and Jackson relates an instance from the experience of Langdon Down in which a speechless idiot during the delirium of fever spoke freely.

When the aphasic has habitually spoken more than one language, certain words may remain in each language. Thus, a woman under my own care would frequently answer "no" as the only English word at her command, but would express her emotion by "Gott in Himmel," the sole remaining fragment of her native tongue.

Functional Aphasia.—Although commonly an aphasia is due to an organic lesion, yet it may be purely functional. It is notorious that under great excitement the power of speech may be lost. In the *Bost. Med. and Surg. Journ.*, December 17, 1885, is an account of a case in which aphasia was produced in a child by fright. In hysteria aphonia is much more frequent than aphasia, and yet undoubtedly there is an hysterical aphasia which may remain for many months, if not years, although not dependent upon any gross lesion of the brain. Dr. Haertz (*Ziemssen's Cyclopædia*, vol. ii. p. 601) states that a paroxysm of aphasia may replace the ordinary symptoms of malarial poisoning. After epileptic attacks there is sometimes a temporary aphasia. The symptom has also been noted in a large number of acute diseases, but probably in the majority of these cases it has rested upon a distinct lesion. It is certainly capable of being produced by reflex irritation. Prof. Bernhardt affirms that in children indigestions, entozoa, and psychical irritations occasionally produce aphasia (*London Medical Record*, October 15, 1886). Kussmaul states that cases have been reported in which collections of fæces in the large intestine or lumbricoid worms have been supposed to be the cause of an aphasia which has disappeared on the expulsion of the irritant. In a case which came under my notice several temporary attacks of aphasia were the result of an overloaded stomach, and were relieved at once by vomiting.

Functional aphasia can usually be distinguished from the organic affection by its temporary or paroxysmal character, but in hysteria the loss of speech may persist for a great length of time. Under these circumstances the positive diagnosis may long be impossible, although the nature of the aphasia may be surmised from the hysterical history of the patient and the absence of evi-

dences of serious organic brain-disease. The sudden recovery of such a case would decide its nature.

Lesions of Aphasia.—In 1836, Dr. Dax first made known at Montpellier that in organic aphasia the left cerebral hemisphere is at fault. Since his earliest publication a large number of cases have been reported, which prove that in the great majority of instances it is the left hemisphere that is diseased: thus, out of two hundred and sixty cases of aphasia collected by Dr. Seguin, of New York (*Quarterly Journal of Psychological Medicine*, January, 1868), in two hundred and forty-three there was right hemiplegia and in seventeen left hemiplegia, the proportion being as 14.3 is to 1.

The connection between disease of the left hemisphere and aphasia is even closer than is indicated by these figures of Dr. Seguin, for there is reason to believe that in a considerable proportion of the cases in which left hemiplegia has been associated with aphasia there have been two lesions. The most satisfactory explanation of this connection yet given is the superior development of the left cerebral hemisphere, due to the habitual excessive use and training of the right hand, which acts not only upon the individual, but, from the laws of heredity, upon the race, the perpetual training of generation after generation resulting in an habitual excessive development of the centres presiding over the right hand,—*i.e.*, of the left cerebral hemisphere. Especially is the habitual act of expressing thought in writing with the right hand believed to lead to the great development of the speech-centres in the corresponding brain-region. In a certain proportion of cases the human individual is born with a superior development of the left hand, or, in common parlance, is left-handed. In such persons it must be acknowledged that the right cerebral hemisphere is the most highly organized. If the theory which has just been enunciated be correct, we should expect to find that aphasia in left-handed people is habitually associated with left hemiplegia.

Drs. Pye Smith, Hughlings-Jackson, and John Ogle have reported a number of such cases. (See Kussmaul, p. 740.) In a very extraordinary case reported by Wadham, a young man, who wrote with his right hand, but was, like his brother, in other respects left-handed, suffered with left hemiplegia with aphasia. It

would seem, therefore, that he used the left hemisphere in writing and the right in speaking. In this case, according to Dr. Bateman (see *London Lancet*, April, 1880), the lesion found after death was a complete destruction of the island of Reil on the right side, the left hemisphere of the brain being healthy. The association of aphasia with left hemiplegia is usually dependent upon its occurrence in left-handed people, but this explanation will not suffice for all cases. Thus, Dr. Michel Catsaras (*La France Méd.*, 1884, vol. ii.) reports a case of complex aphasia with left hemiplegia in a man who was not left-handed. Hughlings-Jackson (*London Lancet*, April 24, 1880) has recorded a similar case. I have not been able, however, to find any case of the character just spoken of in which there has been an autopsy, and the possibility remains that in these cases a double lesion has existed. Even if, however, an exceptional case should be clearly made out, it would have to be viewed simply as an exception to the general law, that the speech-centres of the left hemisphere are active, whilst those of the right hemisphere are dormant.

The connection between the frontal lobes and word-thought was originally pointed out by the celebrated Prof. Goll in 1825. His pupil Bouillaud located the speech-centres in the divisions of the cerebrum over the fissure of Sylvius and in front of the fissure of Rolando. In 1861, Broca affirmed that the integrity of the left third frontal convolution, and perhaps also of the second, is essential for the development of articulate speech. The cases of organic aphasia which have been reported in the last twenty years are far too numerous for analysis here. They undoubtedly, however, show that the third frontal convolution and the island of Reil in the left hemisphere are closely connected with the speech-function. The conclusion reached by Kussmaul is that the left frontal lobe, and especially the third frontal convolution, possess by no means a monopoly of aphasia, although it is most frequently brought about by lesions at this point. The island of Reil comes next in frequency to the frontal lobe.

It seems to me that we must consider it proved that permanent complex cortical aphasias are due to disease of the third frontal convolution or the island of Reil in the left hemisphere, and that in this position are located the centres of word-thought. Kussmaul states that he has been able to discover only two ob-

servations in literature in which a lesion of the third left convolution has occasioned no aphasia, and that in the reports of these cases it is not stated whether the patient was right-handed. It has not been the habit of most observers to examine microscopically the convolutions of the island of Reil in cases of aphasia without apparent lesion, and without such examination no weight can be attached to a case in which aphasia has existed without lesion of the speech-convolutions. In one case of my own, in which to the naked eye the speech-convolutions were healthy, the microscope showed that their vessels had undergone degeneration and the cells atrophy. I have also had opportunity to examine specimens from an unreported case which occurred in the practice of Dr. A. V. Meigs, in which the only gross lesion found was a large patch of softening in the neighborhood of the left corpus striatum, but in which the microscope showed that the blood-vessels and ganglionic cells of the third frontal convolution were profoundly affected, there being even minute patches of softening and microscopic hemorrhages. In the light of such cases as these the scientific accuracy of much of our aphasic literature becomes very doubtful, and I do not think that there is a properly-observed case on record in which a permanent aphasia has existed and the third frontal convolution region been normal. If such a case should be reported, the aphasia would have to be considered as due to an anatomical variation or as produced in some indirect way. It is well known that the effects of gross lesions of the brain often extend far beyond their immediate confines, and if a gastric irritation may inhibit the action of the speech-centres, it is not strange that a tumor or softened mass of brain-tissue may sometimes have a similar power. It must also be remembered that aphasia should be produced by lesions of the white matter which interrupt the passage downward of the fibres from the speech-centres.

In regard to word-blindness and word-deafness, the number of autopsies which have been made is not as yet sufficient to allow us to consider the conclusions reached as fixed. The centres for sensory aphasia are located by Wernicke along the margin of the fissure of Sylvius in the first temporal convolution. The region of sensory aphasia is, however, probably a much wider one. Grasset (*Montpellier Médical*, 1884, p. 52) makes three central positions for the aphasic alterations:

First. Centres of verbal deafness in the first left temporal convolution.

Second. Centres of verbal blindness in the inferior parietal lobule.

Third. Centres of transmission, or ataxic aphasia, at the foot of the third temporal convolution.

Dr. Amidon (*New York Medical Record*, November 15, 1884) collected twenty-four cases of sensory aphasia: in eight, with lesions affecting the visual and auditory regions of the left hemisphere, there were both word-blindness and deafness; in two, in which the area of vision alone was affected, there was word-blindness; in fourteen, with the lesion in the auditory region, there was word-deafness. Wernicke locates the lesion in paraphasia in the medullary tract connecting the sensory aphasic region with the island of Reil. In a case reported by Dr. S. G. Webber (*Boston Medical and Surgical Journal*, December, 1883) the hemorrhage was situated so close to this tract as to act readily upon it by pressure. In this case the recovery of the speech-function during life showed that the effect of the lesion was temporary, and therefore probably an indirect pressure-effect.

The course of the fibres which run from the speech-centres of the frontal lobe is not known; undoubtedly, however, a clot in the neighborhood of the claustrum, which does not directly implicate the centres, will produce aphasia, probably by dividing conducting fibres and isolating the centres. I have made autopsies upon two such cases, and Drs. Farge, Popham, and Jaccoud have each reported similar instances.

The lesion of aphasia varies greatly in its nature. It may be a clot, a tumor, an abscess,—indeed, any form of acute or chronic localized alteration of the brain-substance. Very frequently it is a narrowly-defined, syphilitic, gummatous meningitis. The arterial supply of the convolutions is received through terminal branches which pass through the pia mater and do not anastomose: hence an excessive thickening or inflammation of the membranes may so interfere with the circulation in the cortex as to affect its function. In this way are to be explained at least some of the cases of aphasia without obvious lesion of the temporal convolutions.

In a large proportion of the cases of aphasia the lesion is

embolic. The region of the brain involved is supplied by the middle cerebral or Sylvian artery, the line of whose course so corresponds with that of the carotid arteries that the blood-current is very likely to carry into it any foreign matters which may reach the brain. The Sylvian artery on the surface of the island of Reil divides into four branches: of these, the first is distributed to the outer portion of the orbital surface of the hemisphere and the adjacent inferior frontal convolution; the second supplies the chief part of the second ascending convolution; the third passes through the fissure of Rolando to the remainder of the ascending frontal convolution, to the ascending parietal convolution, and to the inferior part of the superior parietal lobule; the fourth branch, lying in the posterior part of the fissure of Sylvius, supplies the inferior parietal lobule and the superior temporo-sphenoidal convolution.

The three speech-centres of Grasset, although conjointly supplied by the middle cerebral artery, are reached by distinct branches of this artery, so that whilst an arterial lesion of the main trunk involves all the speech-centres and gives rise to a complex aphasia, a lesion of one of the branches may involve either of the aphasic regions separately and give rise to one of the special forms of the affection.

In children aphasia sometimes exists without hemiplegia. It may be due to an arrest of development, which, I believe, may be caused by an emotional storm. A case which died under my care had the history of the child's having been well and strong until it was nearly two years old, at which time it was beginning to talk successfully. It was then taken into a railway-train, was excessively frightened, and screamed for two hours. From this time it ceased to talk, developed convulsions, and, after several years, died. At the autopsy the only lesion I could find was complete failure of development of the convolution of the left island of Reil. The brain looked as if this had been abruptly gouged out of it.

GENERAL MEMORY.

Like most functions of the organism, memory may be stimulated, perverted, or depressed.

Exaltation of Memory.—A distinct, indisputable stimulation

or increase of the memory under the influence of pathological processes is a phenomenon rarely to be distinctly recognized. Forbes Winslow details cases in which an extraordinary excitation of the memory and of other mental functions preceded an attack of apoplexy; and it is probable that in the mental exaltation which precedes a general mania or occurs in the peculiarly dangerous form of insomnia due to excitation of the cerebral cortex the memory may share in the general functional excitement of the brain.

Failure of Memory.—Loss of memory is an exceedingly frequent symptom of organic brain-disease. It cannot be said to be characteristic of any particular form of brain-disease, but is liable—indeed, almost certain—to occur in organic affections of the cerebral cortex. Its diagnostic importance comes from the fact that, unless due to obvious acute disease or connected with insanity, it is a strong indication of an organic affection of the brain. A degree of failure may, however, arise from simple brain-exhaustion.

Usually the patient or his friends recognize even a slight loss of memory; but sometimes very careful search is required for its discovery. Under these circumstances the physician must question the patient as to the small events of the last twenty-four hours, and not be misled by that vividness of recollection of the long past which sometimes causes the sufferer to declare that his memory is even stronger than normal. In doubtful cases of general paralysis of the insane, failure of the memory is of special value in enabling us to distinguish the organic insanity from functional mental disturbances which may simulate it. According to my own experience, failure of memory which is not accompanied by paralysis for the time being of all the functions of the mind, as in insanity, is of serious import in proportion to its completeness.

CORRELATED DISORDERS OF MEMORY AND CONSCIOUSNESS.

As has already been stated, a memory is possessed by all varieties of ganglionic nerve-cells, but that intellectual function to which the name is usually restricted is so closely related with consciousness that we can scarcely conceive of its existence without consciousness: nevertheless, the connection of memory with dreaming shows that it is a separate function from consciousness.

There are a good many reasons for believing that the impressions of all events with which an individual has been connected are indelibly recorded upon his brain-tissue, although he may not be able to bring such impressions into conscious perception. At the approach of death, or under the stimulation of disease at a time when consciousness is wanting, persons will frequently speak in foreign tongues, recite passages of prose or poetry long since forgotten, or give detailed accounts of events that occurred in their earliest childhood and of which they have in their normal condition not the slightest remembrance. It would therefore appear that two distinct functions or acts are involved in conscious memory,—one the preservation of the records, the other the dragging out of such records into the light of consciousness and their recognition by the personality of the man. In certain diseases when consciousness is obliterated the connection between the stored records of the cerebral cortex and the automatic speech-centres is so close that the latter act in obedience to the records, and the unconscious patient speaks in an unknown tongue, or relates occurrences of which he has no conscious memory.

When the link that binds consciousness to memory is broken by disease, consciousness may exist without memory. Under these circumstances consciousness is isolated from the past, although the past may still be connected with the present by an automatic unconscious memory.

This is illustrated by the case (Ross, *Nervous Diseases*, vol. ii. p. 880) of a man who was wounded during the Franco-German war in such a way as to lay bare the brain for about two and a half inches in the left parietal region. As the result of this he was subject to attacks lasting from twenty-four to forty-eight hours, in which, although in a condition of apparent partial consciousness, he had no sensitiveness of any part, and was unaware of physical pain. Nevertheless, his will was at once influenced by external objects. If set upon his feet, the contact with the ground started him to walking, when he marched straight on, quite steadily, with fixed eyes and without saying a word. If he met with an obstacle, he would touch it and try to make out what it was, and then get out of its way. A pen placed in his hand started him to writing. Dr. Ross says of him,—

"Give him cigarette-paper and he will take out his tobacco, roll a cigarette, and light it with a match from his own box. But ignite a match yourself and give it him, he will not use it, but let it burn between his fingers. If his tobacco-box be filled with any trash, he will roll his cigarette and smoke without perceiving the hoax. If a pair of gloves be put into his hand, he will put them on, and, being reminded of his profession, will look for his music. If a roll of paper then be given to him, he will assume the attitude of a public performer and begin to sing."

Loss of Personal Identity.—An attribute of the human understanding which is dependent upon the existence of memory and consciousness is the sense of *personal identity,*—*i.e.*, the conviction of the individual that he is the same person as he has been in the past. The unbroken chain of events recorded from an indefinite past correlated with the consciousness of the present gives the realization of the unity of the present with the past. This sense of personal identity is destroyed by a complete loss of memory, which loss may be abrupt and be unaccompanied by impairment of consciousness or of rationality. I have seen this association of symptoms continue for several days after a sunstroke, so that the patient, who had been brought by ambulance into the hospital, was unable, after he had recovered his mental faculties and was perfectly rational, to give any clue to his personality which could lead to his identification.

Double Personality.—Hasheesh and perhaps some other drugs have the power of producing a sense of *double personality,*—a condition in which the subject feels as though he were two distinct personalities, one holding intercourse continually with the other. In insanity this feeling of double personality may be the basis of delusion. Such delusion usually takes the form of an absolute belief in a dual existence: thus, in a case of my own, an insane man believed that he and all others of the human race had their "doubles," which were not to be distinguished from their proper personalities. The life of the patient was overwhelmed by the constant fear that he was not himself, but his own double.

Double Consciousness.—Double personality is to be distinguished from the extraordinary phenomenon to which the names of *double consciousness, periodic failure of memory,* and *periodic amnesia* have been given. In this state there is undoubtedly a disorder

of memory, but in most cases all the intellectual functions are deeply involved. Before attempting an analysis of double consciousness I shall briefly sketch some of the more important recorded instances. The earliest record of such a case that I have been able to find is by Dr. Mitchell (*Med. Repos.*, p. 185, New York, 1817). A very highly educated young woman fell without warning into a deep sleep, which lasted for many hours. On waking, she had lost all her former knowledge; her memory had become a *tabula rasa*, every trace of her past culture having disappeared. It was necessary for her to relearn everything. After extreme effort she became familiar with surrounding persons and things, acquired the alphabet, then learned to read, then to write, and finally to reckon. Some months later she again fell into a deep sleep, and awoke in her normal state. She now knew all that she had learned in her original condition. For many years after this she alternated between the first and second conditions, in each state knowing only what she had learned in the previous periods of the same state. When she made acquaintances she recognized them again only when she was in the state in which she had been at the time of the first meeting. Her handwriting, which was very good in her first condition, was very bad in her second state.

Dr. Azam (*Annales Méd.-Psych.*, 1876, vol. xvi.) reports a case of double consciousness occurring in an hysterical girl. In this patient the change from one state to the other was always preceded by a profound sleep lasting three or four minutes, this period of repose being ushered in by an intense headache. In her abnormal state the girl was extremely gay and vivacious, and remembered perfectly all that had passed both during previous similar abnormal conditions and during her normal life. There was no delirium, no hallucination, no false appreciation, but the intellectual faculties were more developed than during her normal condition. After this condition had lasted a variable length of time, her gayety suddenly disappeared, her head dropped, and she fell into a deep sleep, out of which she awakened in a condition of great sadness. She forgot all those things that had happened during her abnormal period, but remembered perfectly everything that she had known or that had been done during the preceding normal states. Having been seduced and become preg-

nant during one of her abnormal periods, she was entirely ignorant of the affair during her normal state, although fully aware of it during successive abnormal periods. Finally, while in her normal condition she was made to understand that she was pregnant, when she was seized with violent hysterical convulsions. After the birth of the child she had no mental trouble for several years, and was married. Somewhat later, after a very painful and exhausting accouchement she had hysterical lethargy, followed by ecstasy and violent hysterical manifestations. At thirty-two she was the mother of a family, and an active business woman. The child which had been conceived during an abnormal period was very intelligent, and an excellent musician, but of a highly nervous temperament, and liable to nervous attacks.

A case somewhat similar to this is reported by Dr. James Mayo (*London Medical Gazette*, vol. i., 1845). A young girl passed repeatedly through two alternating different states of mental existence. During the abnormal periods she was extremely excitable, and had mental attributes much above her normal condition. She made progress in needle-work and in intellectual acquirements far beyond what was possible with her natural talent. She also became very vivacious in conversation, but did not recognize her relations to her father and mother, calling them by wrong names. On the subsidence of her abnormal state her recollection of kindred and friends returned, and she resumed her quiet, dull character. In the abnormal state she remembered without the slightest confusion all that had happened in previous abnormal periods, and what she had learned either manually or intellectually, but knew nothing of what had occurred in her normal conditions. In her normal conditions she had no knowledge of anything that had happened or of anything that had been learned during the abnormal states.

I have seen one case which offered symptoms resembling those of double consciousness. They were produced by a depressed fracture caused by a blow upon the head. Previous to trephining there were at least four of the abnormal states, but since the operation, over a year ago, there has been no return, although the patient's mental condition is not good. In the first of his spells he went to a railroad-dépôt, bought a ticket, travelled on the cars two or three hours, and, after getting out, met an acquaintance,

who expressed great surprise at seeing him, and asked why he had come, receiving the reply that he had come to try to get work. The man then went to a friend's house to dinner, where he talked and ate, until suddenly he waked up, with an inquiry of intense surprise as to how he had got there. He appeared to have no recollection of his trip, except of a few minutes, his remembrance of which was so vivid as to indicate that he had then had a waking spell. During the abnormal period the man's behavior suggested to his acquaintances only that he was distraught. I was unable to obtain evidence that he exhibited during the spells any memory of acts performed in previous similar periods.

It does not seem necessary to abstract any more of the few recorded cases of double consciousness for the purposes of present illustration. In a typical case there is, first, an abrupt loss of memory at the beginning of each paroxysm for everything that has happened during paroxysms not of the same series; secondly, a change in the personal character of the individual, the disposition, the habits of thought, and even the intellectual powers being altered.

Double consciousness may exist in various degrees. Thus, in a case reported by Dr. Samuel Jackson (*Amer. Jour. Med. Sci.*, 1869, p. 18), the character was affected rather than the memory. An hysterical young lady was attacked with nausea and vomiting, followed by a complete alteration of character and change of voice. Formerly mild and gentle, she became abrupt and rude, and yet would so perform her household duties that it was often difficult to determine in which condition she was. The attacks came on daily, without apparent cause. The symptoms in this case seem to represent only an exaggerated form of moodiness; and there can be no doubt that even the most complete double consciousness is closely related on the one hand to epilepsy and on the other to hysteria and to insanity. In my case detailed above, the cause of the attack was a blow upon the head,—a not rare cause of epilepsy,—and there was no binding memory between the spells. In that form of epilepsy in which there are automatic movements, and also in cases of epileptic delirium, the paroxysm is in many ways related to the second period of double consciousness, but differs in that the individual does not preserve any reasonableness or capability of being affected by other persons,

and does not remember what has occurred during previous spells of similar type. On the other hand, in a large proportion of the cases, double consciousness has occurred in hysterical women, and the phenomena pass almost insensibly into those of hysterical semi-consciousness and delirium.

In insanity there is often a change in the whole manner of thought and character of the individual. In a case of melancholia long under my care, the patient after recovery was subject to frequent attacks of transitory melancholia, which lasted from a few to many hours: whilst perfectly contented and happy, she would say, "It is coming," and in a few moments would be covered over as it were with a wave of emotional depression which would, for the time being, completely alter her habits of thought and her behavior. In a case of profound apathetic melancholia recently at Burn Brae Asylum, near this city, the patient one day suddenly said, "I have had a revelation: my sins are forgiven me," and, after weeks of absolute voicelessness, became talkative, rational, and active in all his sympathies, without, however, being unduly excited. Not long after this the man relapsed into his insane condition. It is said that similar sudden changes have previously occurred several times. This man appears to carry his memory over from one state to the other, and in this only do his symptoms differ from those of typical double consciousness. If in such a case of insanity as this the continuity of memory should be broken, there would be a typical double consciousness.

The close relation between insanity and double consciousness is further illustrated by a case reported by Dr. David Skae, in which a man after eighteen months of typical melancholia developed a twofold life, being on alternate days sane and insane. On melancholic days he neither eats, sleeps, nor walks, but sits incessantly turning the leaves of the Bible and complaining piteously of his misery. At this time he has no remembrance of the days in which he is well, nor of any engagements made during them: he does not, and cannot be made to, recognize the existence of such days, but contemplates the future with hopeless despondency. On the alternate well day he denies that he has any cause of complaint, believes that he was well the previous day, transacts business, takes food and exercise, and is entirely free from delusions or despondency. He also anticipates no

return of his illness, and has no memory of his bad days. He remembers exactly the transactions of his previous well days, and persists in making business engagements for the following day,—*i.e.*, for his melancholic day,—although repeatedly assured that at the time named he will be unable to attend to business.

There is a very rare mental condition known by German writers as *Doppeltwahrnehmung*, or *double perception*, which is liable to be confounded with the condition I have just described under the name of double consciousness. The peculiarities of this affection are well portrayed in the report by Dr. M. Ruppert (*Allgemeine Zeitschrift für Psychiatrie*, 1869, vol. xxvi. p. 531) of a case in which whenever the man read to himself he would plainly hear each word repeated as though a chorus of fifty or sixty female voices were speaking to him, and when he ceased to read he would hear the last words read after him. This reading after him disappeared as soon as he spoke aloud, and was prevented by his reading aloud. In one sense of the term consciousness, this patient had a double consciousness; but it is plain that his symptoms were much more closely related to hallucinations than to the state commonly known as double consciousness.

CHAPTER X.

DISORDERS OF CONSCIOUSNESS.

In a book like the present an elaborate discussion of the so-called physiological theories of sleep would be out of place. It seems to me, however, that a few words upon the subject are required. According to some physiologists and neurologists, natural sleep is induced by the withdrawal of blood from the brain. It must be allowed that the concordant results of experiments show that during sleep there is a more or less pronounced cerebral anæmia, which on awaking is replaced by turgescence of the cerebral vessels. This is not, however, proof that sleep is induced by the withdrawal of the blood. It is a universal law that cessation of functional activity is immediately followed by lessening in the amount of blood in the part. I conceive, therefore, that the sleep is the cause of the bloodlessness, and not the bloodlessness the cause of the sleep. This is certainly in accord with clinical experience, which to my mind proves that insomnia may be connected either with excessive anæmia or with excessive congestion of the cerebral cortex. Thus, the wakefulness of anæmia is well known, as is also the insomnia of acute mania. Some physiologists have attempted to explain the production of sleep by supposing that certain chemical compounds are formed during the activity of the day, which, circulating in the blood of the brain, act as hypnotics on the cerebral cells. There is no evidence worthy of attention establishing any theory of this character, and to my thinking such chemical theories are upon their face so improbable that they should be received only after the clearest proof. Concerning sleep, as concerning other functions of the human organism, the simplest explanation is the most probable. In the greater portion of the active tissues of the organism rest alternates with activity, and the brain in its sleep conforms to this general habit. The best explanation of sleep, then, is that when exhausted by effort the cortical brain-cells pass into a condition of functional inactivity, during which their power of further effort is

recuperated. Because consciousness is the expression of functional activity in these cells, therefore when these cells do not exercise their function there is unconsciousness,—*i.e.*, sleep. That sleep, or functional rest, should be more or less periodical seems essential from the very nature of the case. Throughout a healthy nervous system a tendency to periodicity of action is marked. In disease this tendency becomes even more apparent. Pain-storms recur with more or less regularity, habits of periodical discharge, at first accidental, become fixed, convulsions develop at intervals, etc. The daily rhythm in the production and dissipation of animal heat during health is a forerunner of the marked diurnal swing of temperature so common in fevers.

The bearing upon the practice of medicine of our belief as to the immediate causation of sleep is very apparent. If we think that insomnia is due to cerebral anæmia, we must treat the anæmia to remove the insomnia. Out of such error have grown other theories, which, though absurd, have been largely dominant. A notable example of these is the belief, at one time wide-spread, that bromide of potassium produces sleep by affecting the blood-vessels. Again, largely in order to sustain their theories, certain neurologists have distinguished sleep, stupor, and coma as essentially diverse conditions readily to be diagnosed in the sick-room. These states are, however, simply the outcome of different degrees of completeness in the suspension of the functions of the cerebral cortex: such a suspension finds its lightest expression in a doze, and its profoundest development in a coma. No rules of diagnosis can be laid down which will enable us to draw any practical lines, sharp and fixed, between the lightest slumber and the most complete unconsciousness. Nor is the unconsciousness of anæsthesia an isolated thing. It is simply a suspension of cerebral function in which a chemical agent is the cause of the paralysis. In the sick-room every grade can be found between light and heavy slumber, between heavy sleep and stupor, and between stupor and coma. For the purposes of discussion we must, however, arbitrarily separate these states. I would define these terms as follows: *sleep* is that condition of unconsciousness in which the subject is readily aroused, and when aroused is easily kept awake by ordinary external stimulations or by his will-power; *stupor* is that condition in which the subject is aroused with great

difficulty, and when left to himself relapses into unconsciousness; *coma* is that state in which it is impossible by external irritation to restore consciousness.

In the present chapter I propose to treat of, first, sudden loss of consciousness; secondly, sleep, its abnormalities and accidents.

SUDDEN LOSS OF CONSCIOUSNESS.

Sudden loss of consciousness is a symptom of such varying import that it is scarcely susceptible of scientific arrangement in a treatise like the present. Nevertheless, it is one of such practical importance as to demand discussion. A blow upon the head, or even upon a distant part of the body, may produce immediate insensibility; but the study of such cases belongs to surgery, and I shall in the present consideration of the causes and collateral symptoms of sudden unconsciousness omit traumatism and its results. Loss of consciousness is an essential part of the epileptic convulsion; but, as this convulsion has already been studied in detail, I shall at present consider only affections in which there are no convulsive symptoms, or in which if the convulsion occurs it is not an essential feature of the disease, but an incident of the attack. Thus, in epilepsy the convulsion is an essential feature, but in a cerebral hemorrhage or a sunstroke it is not of such character; and if a convulsion occur in an apoplexy it belongs to the individual case, because it is not necessarily present in the disease.

The ordinary non-traumatic causes of the sudden loss of consciousness are epilepsy, hysteria, syncope, sunstroke in its various forms, apoplexy, various forms of poisoning, and malignant systemic diseases.

The symptoms which attend the *hysterical* and the *epileptic* loss of consciousness, and the methods of recognizing the nature of such attacks, have been thoroughly discussed in a previous chapter. *Syncopal* loss of consciousness, due to a failure in the supply of blood to the brain, is to be recognized by the extreme pallor of the subject, by the absence of the pulse, or its excessive threadiness or weakness, at the wrists, and by the greatly enfeebled action of the heart, as shown by the weakness of the cardiac sounds and impulse. Its diagnosis is facilitated by noting that it occurs after

exertion or during great excitement, and in a subject already enfeebled by disease, hemorrhage, or accident, or else of a naturally weak constitution.

Sunstroke.—During the summer months sunstroke is a very frequent cause of sudden loss of consciousness. It develops only after exposure to heat, either natural or artificial. It may happen in the day or in the night, and is especially fatal in sugar-refineries and other places where the heated air is saturated with moisture. It occurs most frequently in unacclimated races, and is very rare in negroes, Hindoos, and other tropical peoples. There are two distinct forms of it,—one of which may be known as thermic fever, the other as heat-exhaustion.

The animal organism is constructed to run upon a certain plane of heat, and whenever this level is departed from all the functions of the body suffer. The nervous system is the most susceptible portion of the organism, and therefore the nervous symptoms are always prominent when there is a great disturbance of the bodily temperature. It appears to make little difference whether the temperature be elevated above or depressed below the norm, so far as the nervous system is concerned. In either case, if the departure from the norm be sufficient, consciousness is lost, the lower brain-functions are implicated, so that the respiratory and cardiac action both become irregular, and death occurs at last usually from paralysis of the respiratory functions. In thermic fever the temperature of the body is greatly elevated. In heat-exhaustion the bodily temperature is depressed.

Heat-exhaustion is very often felt in a mild degree by feeble workers in hot weather. There is a sense of weariness and distress, pallor of the countenance, failure of the muscular force, and finally failure of the pulse. Severe examples of the affection are rare. In these the symptoms may develop almost as suddenly as in thermic fever. Thus, in a case brought into the Centennial Hospital during my service in 1876, there was sudden unconsciousness, with muttering delirium; great restlessness; a facial expression of collapse; profuse perspiration bedewing the whole surface; rapid, feeble, scarcely perceptible pulse; and a mouth-temperature of 95°. Although in their general aspect the symptoms of heat-exhaustion resemble those of thermic fever, the true character of the case should be at

once recognized on touching the ice-cold surface of the body. The only disease or condition readily confounded with heat-exhaustion is collapse from other causes. I have seen cases of internal aneurism, of pernicious malarial fever, and of other affections picked up in the streets and brought into the hospital in collapse in which a mistake in diagnosis would have been very excusable. If such a case should happen upon an intensely hot day, and the bodily temperature be much below the norm, the diagnosis might have to be reserved. Except as regards pernicious malaria, however, this is a matter of little importance, because the treatment of heat-exhaustion is the same as that of collapse with lowered temperature from other causes.

Thermic fever, *heat fever*, or *coup de soleil* (sunstroke) usually comes on without distinct prodromes, although frequently there is a great sense of distress or of a general burning heat before the loss of consciousness, which may also be immediately ushered in by chromatopsia, or colored vision,—the whole landscape being deluged in a blue, yellow, or red light. The unconsciousness ordinarily develops abruptly, and is complete, although very frequently it is associated with muttering delirium. There is usually great muscular restlessness, which in some cases becomes convulsive or is replaced by violent epileptiform convulsions. Sometimes the patient is profoundly relaxed and quiet. The surface of the body, at first dry, often later in the attack gathers upon itself an excessive perspiration, which does not, however, reduce its burning heat. The face is flushed, and the eyes are suffused. The rapid pulse is sometimes bounding and apparently strong, although almost invariably compressible; frequently it is feeble and even thready, especially if the symptoms have lasted for some hours. Vomiting is very common; purging is in bad cases almost always present. The whole body is apt to exude a peculiar odor, which is especially strong in the fæcal discharges. The characteristic symptom is the high temperature, which, as measured in the mouth or rectum, may reach 112° or 113°, and is rarely below 108° in cases severe enough for unconsciousness to be present. The urine is scanty, sometimes albuminous, not rarely finally suppressed. The breathing is more or less labored, and often irregular, and towards the last generally becomes more and more shallow. Although at times the patient suffering from

thermic fever may be partially aroused by shouting, shaking, etc., the unconsciousness is often absolute. The pupils are variable, sometimes contracted, sometimes dilated.

The diagnosis of this form of thermic fever is usually free from difficulty: the known exposure to heat,—*i.e.*, to the cause of sunstroke,—the unconsciousness, and other symptoms, and the very high temperature both of the surface and of the interior of the body, are characteristic. If, however, an apoplexy should occur upon a very hot day and be, as it might, associated with a sudden rise of temperature, the diagnosis would not be easy; indeed, if the mouth were not drawn and the general relaxation prevented the recognition of hemiplegia, the diagnosis might be impossible.

In this country the profession has been accustomed to recognize as sunstroke, or thermic fever, only the severe cases which approach to the symptoms that have just been detailed. For many decades, however, medical practitioners in India have known that there is a form of continued fever due to heat which, under treatment, may gradually subside, or which may at any time end in a sudden explosion like that of true sunstroke. Dr. John Guitéras (*Therapeutic Gazette*, March, 1885) has shown that this mild form of thermic fever occurs in the subtropical portions of the United States. These cases have usually been supposed to be instances of typhoid fever, from which, according to Dr. Guitéras, they are to be separated by the suddenness of their onset, the temperature of the first day reaching 103° F., or even higher; by the course of the fever, which is extremely irregular, indefinite as to duration, and almost always has the morning remission more accentuated than in typhoid fever; by the absence of petechia, miliaria, rose-colored spots, tenderness, and gurgling in the iliac fossa; by the tongue remaining moist, with only a light creamy coating, interrupted by a clear streak along the edges and median line; by the absence of cerebral symptoms, except at times when the fever rises very high; and by the mental alertness instead of hebetude. Diarrhœa, although often absent, may be present, and even be severe and bloody.

Many cases of obscure indisposition during intense hot weather, especially in children, are really mild instances of derangement of function of the body by heat,—that is, of thermic fever. Dr. C. Comegys, of Cincinnati, was the first to call attention to

the fact that cases of *cholera infantum*, so called, are frequently instances of thermic fever and yield at once to the use of the cold bath. It has long been known that in cholera infantum there is often a sudden increase in the fever, with or without lessening in the number of the passages, but with a rapid loss of consciousness, which is likely to end in death. Under these circumstances the cause of the cerebral symptoms is the elevation of the temperature. As it occurs in our large cities in hot weather, cholera infantum is frequently, if not usually, a form of thermic fever, and yields with great readiness to the systematic use of the cold bath. In all cases of this character the physician should use a thermometer, and if the temperature be found distinctly above the norm it should be reduced by systematic cold bathing. (See article on "Sunstroke," *Encyclopædia of Medicine*, vol. v., Philadelphia, 1886.)

Apoplexy.—The term apoplexy, as used in this work, applies to cases in which consciousness is suddenly lost as the result of disturbance in the circulation of the brain. Scientific accuracy would require that cases of arrest of the cerebral circulation should be separated from cases of congestion or hemorrhage: the diagnosis, however, between loss of consciousness which is the result of arrest of circulation in the brain by an embolus and the unconsciousness which is due to a sudden rupture of a vessel and hemorrhage into the brain is frequently not possible. Moreover, it is by no means certain that even in hemorrhagic apoplexy the suspension of cerebral functions is the result of a congestion or of an excess of blood in the brain. For these reasons it seems to me wisest to consider under one heading all losses of consciousness connected with circulatory brain-disturbance, and afterwards to point out the few facts of value that we have as guides in separating between the forms. Again, it should be said that, except by the presence of paralysis or convulsions, we have no way of distinguishing between an apoplexy which is simply due to congestion of the brain and one which is connected with more or less permanent organic changes. It has been denied that there is such a thing as simple *congestive apoplexy;* but there can be no doubt of its occurrence. I have seen it developed without obvious cause, to be recovered from without sequelæ, and never to recur in after-life. A congestive apoplexy may be due to a brain-tumor, or to a

syphilitic disease of the brain or its membranes. For a detailed discussion of these comas, see article on Organic Coma.

For description and study, cases of apoplexy may be arranged in two classes, to which the names of sthenic, or congestive, and asthenic, or syncopal, may be given.

There are no etiological differences between these two classes. Extreme typical cases differ widely and characteristically in their symptoms, although they rest upon similar structural changes. Moreover, every possible intermediate variation occurs in nature between an extremely sthenic and a typically asthenic apoplexy, so that the two classes are not naturally separated from each other, but are artificially characterized for the purposes of discussion.

The apoplectic attack may come on with great suddenness. It may, however, be preceded by prodromes, which are in some cases affirmed to have lasted for some hours. Sometimes without distinct warning the patient drops unconscious, or he may become confused in speech and manner, and then suddenly be stricken, or else gradually grow more and more heavy and finally sink into unconsciousness. At the height of the attack the unconsciousness is complete. The pupil is fixed, dilated or contracted, as the case may be. In the congestive form the face and conjunctivæ are intensely suffused, dark purplish red. The breathing is loud, snoring, and stertorous. The pulse is usually full and bounding. It may be slower or more rapid than normal; occasionally it is small and hard. The surface is warm.

In the syncopal form the face is pale, and the breathing quiet, or, if stertorous, not loud and harsh in its sound. The surface is coolish, and the pulse rapid and feeble.

Paralysis, convulsions, and great rise of temperature may occur in either variety of apoplexy. They are proof of the presence of an organic lesion. The apoplexy may end in death, which is commonly developed by a more or less rapid increase of the symptoms. The unconsciousness remains complete; the pulse, whether originally strong or feeble, continually falls in force; the respiration grows more and more shallow, or more and more irregular, and may at last gradually die away or suffer sudden arrest. The cerebral reflexes are frequently lost early in a case of severe apoplexy, especially when the basal region is invaded by the lesion. Thus it is that the power of swallowing is affected. Complete

loss of this function is a very serious if not a fatal symptom. When recovery occurs it is usually gradual, although in rare cases the patient may arouse himself suddenly. Even when the patient seems rational, mental action may still be very imperfect.

Unconsciousness from Indirect Causes.—Unconsciousness more or less closely resembling that of apoplexy occurs from a multitude of causes. Such unconsciousness is usually developed gradually, and is accompanied by other symptoms which at once distinguish it from the unconsciousness of apoplexy. With a clear history there is rarely any difficulty in distinguishing these various forms of unconsciousness from that due to disturbances of the cerebral circulation. Very frequently, however, in hospital practice the physician is called upon to make immediately such diagnosis as may be necessary for treatment without having knowledge of the antecedents of the attack. The importance and difficulties of such diagnosis seem to me to justify the consideration in this place of the more important forms of unconsciousness which are likely to be brought into a hospital.

In the first place, the patient should always be carefully examined for the evidences of traumatism,—cuts about the head, bruises, etc. I once saw at an autopsy upon a man who had been brought into the hospital unconscious, and in whose case there was no suspicion of traumatism, a knife-blade projecting two inches into the brain, and broken off close to the outside of the skull. A traumatism may be present without the physician being able to detect it, but usually some local indications of the injury will be revealed by close inspection. In the case just spoken of the wound was very narrow and small, and was entirely concealed by the bushy hair.

Loss of consciousness may be caused by poisoning and by great emotional disturbance, or it may be an early symptom of acute blood-diseases. Unconsciousness from acute alcoholism is very frequent in large cities. Not very rarely persons supposed to be only dead-drunk are put into station-house cells, to be found dead some hours afterwards. Even when suspicion is aroused, the diagnosis between apoplexy and deep intoxication may be very difficult. The odor of alcohol about the breath or the person, and its presence in the urine, point towards alcoholic poisoning, but cerebral hemorrhage may occur after either moderate or im-

moderate drinking. It is in such cases that mistakes are most liable to occur. Whenever by shaking, shouting in the ear, etc., a drunken person cannot be aroused, the probabilities of cerebral hemorrhage are strong enough to justify the temporary diagnosis and the institution of proper measures of relief. A very careful examination should be made for evidences of paralysis. Under the conjoint influence of alcohol and cerebral hemorrhage the universal muscular relaxation is so complete that the detection of a local palsy may be a matter of the greatest difficulty. Drawing of the face to one side is of course decisive, and if in breathing the air comes out with a sort of puff and pulling of one corner of the mouth, the case is one of cerebral hemorrhage. If the patient be at all restless, the motionlessness of the arm or leg of one side will usually betray a hemiplegia. If the pupils are unequal, the probabilities are in favor of cerebral hemorrhage. In any doubtful case the physician should be very careful not to affirm that the patient is suffering simply from drunkenness, but should reserve his opinion.

Opium-poisoning produces symptoms very similar to those of apoplexy, and I have seen cases in which the diagnosis was simply impossible. The presence of hemiplegic or monoplegic palsy is, of course, decisive. But I have seen inequality of the pupils produced by opium. The remarks made in regard to distinguishing alcoholic unconsciousness apply with equal force to opium-poisoning in its advanced stage.

Uræmic poisoning is a very frequent cause of unconsciousness, and, unlike alcoholic and opium poisoning, is often associated with convulsions. The diagnosis between uræmic and organic convulsions has been already discussed. (See page 118.) It is sometimes impossible to make an immediate diagnosis between the quiet form of uræmia and cerebral hemorrhage, especially since in uræmia serous effusion into the cerebral ventricles is common. Cerebral hemorrhage itself is not rare in advanced kidney-disease. In every case brought into a hospital unconscious the urine should be at once examined. If it be normal in specific gravity and free from albumen, the probabilities are strongly against uræmia. If the urine be scanty and of low specific gravity, much more if it be albuminous, the patient is probably suffering from kidney-disease. The presence of local palsy, even though there

be advanced renal degeneration, is demonstrative of either hemorrhagic or serous exudation into the brain. A temperature above the normal without convulsions would, according to the teaching of the French clinical school, prove that the patient is suffering from something else than uræmia. I, however, doubt very much the correctness of this teaching. (See page 121.) Usually, but not always, the patient in simple uræmia can be aroused to some slight extent.

A partial unconsciousness may result from various *irritative poisons* which do not commonly affect the cerebrum. I have seen a marked stupor amounting to almost complete unconsciousness the chief symptom of poisoning by Paris green, and there are numerous cases on record in which the ingestion of large doses of tartar emetic or other violent irritant has been followed by symptoms similar to those of my case. In some of these cases when no history is obtainable a correct diagnosis may be almost impossible: nevertheless a trifling circumstance, if the physician be sufficiently alert, may give the necessary clue. The circumstances of the case, the friends of the patient, and, if he be at all sensible, the patient himself, should be most carefully examined.

A very important class of cases is those in which collapse with more or less complete unconsciousness occurs as an early symptom of severe *internal disease*,—either a local affection, such as a gastric or an intestinal perforation, the rupture of an aneurism, etc., or a general blood-disease.

With a clear history the diagnosis may be very easy; but often in practical life the history is misleading or altogether absent. The suspicion of the physician should be aroused by noticing that the loss of consciousness is not complete and is accompanied by evidences of cardiac failure, coolness of the skin, and a peculiar "decomposed" expression of the countenance seen only in mortal illness, and usually known as the Hippocratic countenance. It is impossible to describe exactly this facial expression, but to the experienced eye it is sufficient for the recognition of the gravity of the disease. In any case offering the symptoms just described, very careful examination should be made to detect the existence of a local lesion, and if evidence be found of a perforation or of any sufficiently severe local affection, the diagnosis becomes clear.

When collapse, with more or less incomplete unconsciousness, is

an early symptom of a malignant systemic disease, we are forced to make the diagnosis by the process of exclusion. Usually it can be determined that the attack is due to some blood-disease, although often it is impossible to know the exact nature of such affection. Except in the case of a pernicious malarial paroxysm, an accurate diagnosis is not of great importance, because it has little bearing upon the treatment. It is, however, vital to recognize the true nature of a malignant chill. This can usually be done by excluding other causes of collapse and by paying attention to the following positive indications:

First. The unconsciousness is partial: the patient can be aroused, and when aroused speaks in a feeble, usually whispering, voice.

Secondly. There are evidences of internal congestions, such as almost complete absence of respiratory murmur, vomiting, gastric or abdominal tenderness, increased percussion-dulness over the liver or spleen, suppression of urine, etc.

Thirdly. The temperature is disturbed: it may be distinctly lowered; or in some cases there is a low external temperature with a high internal temperature.

Fourthly. A history of previous malarial attacks or of exposure to malarial influences can be obtained, and the season of the year is usually that at which malarial fevers prevail.

Fifthly. The patient is an adult, and does not present paralytic or other symptoms characteristic of cerebral hemorrhage. In children malignant malarial paroxysms do not occur, at least in this climate (that of Pennsylvania), whilst primary collapse from acute malignant constitutional affections, not malarial, is in adults excessively rare.

In any doubtful case the patient should be given the benefit of the doubt, and when the collapse has been recovered from, fifty grains of the sulphate of quinine should be administered during the next twelve to eighteen hours.

Collapse with more or less complete loss of consciousness may result from *emotional excitement.* Many years ago, during a cholera epidemic, in answer to a sudden professional call, I found a man in collapse and partially unconscious, who, when aroused, said in a suppressed, whispering voice that he had had a sudden furious attack of vomiting and purging, which had ceased but had left him in his present condition. I supposed that the case was

one of cholera; but the sequel proved that shortly before I saw him the man had cut the throat of a prostitute after cohabitation with her and left her dead in her bed. The collapse I witnessed was caused by the emotional reaction which developed when the madness of his orgy had passed off sufficiently to allow him to recognize the results of his actions.

SLEEP—ITS DISORDERS AND ACCIDENTS.

In treating of sleep and its disorders I shall divide the subject into three parts:

First. Abnormal wakefulness.

Second. Abnormal somnolence. Morbid sleep.

Third. Accidents or groups of symptoms which occur during sleep, and which are not elsewhere spoken of in this book.

ABNORMAL WAKEFULNESS.

In cases of *simple insomnia* the form of the sleeplessness varies. In some instances the subject is simply unable, when bedtime comes, to go to sleep. In other cases he goes to sleep readily, but in the course of two or three hours wakes, and is unable to slumber again. The latter form of insomnia, in my experience, is not commonly the precursor of severe mental affection. This form of insomnia is, on the other hand, often obstinate.

Insomnia may be prodromic of various diseases of the brain. It is very common in the insanities. It is also present not rarely in such general organic brain-diseases as general paralysis of the insane, but is seldom a symptom of tumor or other focal brain-lesion. It may be produced by various diseases of organs other than the cerebrum. It may exist, however, in its most aggravated form without other evidences of cerebral disturbance, and in some cases cerebral exhaustion, and even more severe mental symptoms, are without doubt produced by the loss of sleep. The diagnosis of the cause of an insomnia is to be made by exclusion. If other symptoms of cerebral disease are wanting, the condition of the heart and kidneys should be carefully examined, because latent disease of these organs occasionally has sleeplessness for its chief manifestation. When no disease of the brain or other portions

of the organism can be made out, the diagnosis of simple or functional insomnia must be settled upon

MORBID SLEEP.

In studying the phenomena of disordered or abnormal sleep, I shall first consider those disarrangements of the function which occur in acute fevers or other diseases not directly connected with the brain.

Disorders of Sleep connected with Acute Fevers.

It does not seem necessary to occupy space in the present volume with any elaborate description of the various derangements of sleep which occur in acute fevers. Such derangements may consist of morbid wakefulness, or of a peculiar wakefulness with delirium and partial unconsciousness, or of true stupor or coma. In some cases these manifestations depend upon high bodily temperature; they may also be produced by an excessively low bodily temperature. In other cases of fever the disturbances of sleep are probably the result of impaired brain-nutrition, due to the alteration of the blood. An improper aeration of the vital fluid, such as occurs in severe pneumonia, may for a time cause aggravated wakefulness, but more usually it manifests itself in stupor and coma.

In some cases of fever the patient will awaken from a sleep of several hours' duration, and insist that he has never closed his eyes during the time, probably because his dreams have been so vivid that they have conveyed the feeling of wakefulness. To this state the name of *coma-vigil* was given by Chomel in 1834. In 1849 the term was applied by Dr. Jenner to a condition entirely different from that just spoken of. In the coma-vigil of Dr. Jenner the patient lies with his eyes wide open, gazing into vacuity, his mouth partially opened, and his face pale and devoid of expression. The pulse is rapid and thready or imperceptible; the movements of respiration can scarcely be made out; whilst the cold moist skin marks the presence of a deadly collapse. The patient is awake, but is absolutely indifferent to everything that is going on about him. In some cases this state is preceded by somnolence. It is much more frequent in typhus than in typhoid fever, and, according to Murchison, in typhus fever

it is an invariably fatal symptom. I have seen it in typhoid fever in a single case followed by recovery.

Nelavan.—Nelavan, or the so-called *African sleeping disease* (*hypnosis* of Drs. Daguaire and Nicolas), is an acute fever in which excessive somnolence is the most characteristic symptom, and is probably dependent upon the direct action of the poison in the blood upon the brain-cortex. First described in 1819 by Dr. Winterbottom, this affection has of recent years had numerous expositors. It is endemic on the west coast of Africa, but appears to occur epidemically in some of the West India islands. It attacks the negroes especially, but has in a number of instances decimated regiments of French troops. In most cases it comes on gradually, but it may begin brusquely. There is at first a slight frontal headache, with a sense of constriction in the forehead, attended by a mild fever. The vision may at this period be disordered. The gait becomes irregular, and not very infrequently there is a distinct ataxia. Even during the first hours of the headache an intense desire for sleep is manifested. This continually increases until the patient is overpowered by an irresistible somnolence. During the period of sleepiness the strength fails, the spirits are depressed, and there is some fever, but usually neither diarrhœa nor constipation develops, and the forces of the circulation are well maintained. The somnolence when once developed continues to become more and more intense, and the patient gradually sinks into a profound coma, out of which he cannot be aroused. There is at this time complete loss of sensibility. The coma may pass quietly into death; but frequently there are more or less violent convulsions, and in some cases sloughing bed-sores mark the failure of nerve-power. There are no pathognomonic post-mortem lesions, but in 1873 Dr. Macarthy called attention to swelling of the glands in nelavan as characteristic. In this he is confirmed by Dr. Nicolas (*Rev. Méd. de la France et de l'Etranger*, 1880). The disease is very fatal: out of one hundred and forty-eight cases seen by Guérin at Martinique but one recovered (*Thèse*, Paris, 1869, No. 201).

Abnormal Sleep.

The discussion of morbid somnolence is in the present state of our knowledge attended by much difficulty, as it seems impos-

sible to draw the lines between the different varieties of unconsciousness, and it is not always possible to give a clear explanation of the cause of the symptoms. In its simplest form the symptom consists of an excessive drowsiness, which causes the subject to fall asleep at any time during the twenty-four hours, even when in the midst of work or conversation, and leads to his passing many hours daily in bed. When the morbid tendency is a little stronger, the impulse to sleep becomes irresistible, and its influence is so continual that the waking periods may be reduced to only a few hours out of a week. No distinction can be made between the sleep that occurs in many of these cases and that of normal slumber. The cases grade regularly from the person who is simply known as a profound sleeper, to the subject who passes most of his time in unconsciousness. In lighter cases the patient can be awakened out of the sleep, but in the more severe forms of the disorder it is not possible to arouse the sleeper. Here also a regular series of cases exists between the sleep out of which the subject is easily aroused and that out of which he cannot be awakened at all.

In the more prolonged cases of excessive sleep the patient remains in a condition of insensibility for weeks, months, or years. Dr. Guéneau de Mussy reported to the French Academy of Medicine the case of a woman who was said to have gone to sleep in 1808, and to have been still sleeping at the time of the report, January, 1838. During this time the woman remained immovable upon her bed, with her limbs drawn up over her stomach. At first she took food, but soon she ceased to receive any nourishment except the sacrament, which was administered on the first Sunday of every month. As this case appears to rest solely upon the authority of a French curé, and not to have been seen by the doctor himself, the account must be accepted with allowance. There are, however, similar instances in literature. Thus, a case is recorded in the *New York Medical Gazette*, iv., 1853, in which a man slept five years; one in the *Richmond Medical Journal*, 1867, in which a girl slept eighteen years; and the "sleeping girl" of Turville is affirmed to have not awakened for ten years (*Lancet*, June, 1880). Dr. Gaulke (*Vierteljahrsch. für Gerichtliche Medicin*, vol. xxvi., 1877) gives an account of a prisoner who for two years had been in a death-like stupor. Dr.

Blondet details the history of a young woman who slept twelve months. Although the correctness of some of these reports is open to doubt, other cases might be cited, and it cannot be gainsaid that the unconsciousness may last for years, either unbroken or interrupted by brief intervals of consciousness.

Morbid somnolence of a mild type passes by insensible gradations into that condition known by some English authors as *trance*, and usually spoken of by French writers as *lethargy*. Trance, or lethargy, may or may not be ushered in by marked hysterical symptoms, such as immoderate depression or excessive gayety, convulsions, hallucinations, etc. Usually in the course of a very short time the symptoms are fully developed, and the subject is in absolute repose. The face may be red and hot, especially in the first days of the attack, but usually it is pale. The pulse at first may be regular and slow, but after a long sleep it is rapid and feeble; the respirations, generally quiet, may at times become hurried, irregular, and even stertorous. In severe cases the movements of the thorax may be so slight as to be traceable with difficulty. The muscular system, often thoroughly relaxed, may be rigid, and in many cases muscular relaxation alternates with muscular contractions, or even contractures. The eyes are opened or closed; very frequently minute tremors affect both the lids and the eyeballs. The jaws are often set, and sometimes an excess of saliva or even foam gathers about the mouth. In the profoundest cases there is complete anæsthesia of both the common and the special senses, so that neither pinching nor cutting, neither cold nor heat applied to the skin, elicits response. The pupils are usually dilated; they often respond to a powerful light, which, however, calls forth no other signs of life. Sometimes the patient can be readily fed by means of a spoon, but generally in severe cases it is necessary to use the œsophageal tube. Usually digestion is good, but the stools are at long intervals and scanty. The urine is in most cases scantily secreted, and is passed involuntarily. Considering the small amount of nourishment taken, the bodily nutrition is often surprisingly maintained, but in prolonged cases there comes, sooner or later, great emaciation. The bodily temperature may in the earlier parts of the attack be somewhat elevated, but ordinarily it is distinctly subnormal. The awaking is usually, but not always, sudden. During the

course of such a lethargy the subject may pass into a condition which has been mistaken for death. The bodily temperature falls, the respiration becomes so passive that no movement of the thorax or abdomen is perceptible, and, unless a feather or other light object be held to the mouth, breathing may seem to have ceased. The beats of the heart diminish in frequency and in force, so that they become imperceptible even upon auscultation. The face takes on the waxy whiteness of a corpse. The muscular system is in complete resolution, the dilated pupil no longer reacts to light, and even the cornea is filmy as in a corpse. This death-like condition may last for only a few hours, or may continue from one to several days, after which, little by little, respiration is re-established, the pulse returns, and the circulation brings new life into the limbs: after such a crisis the subject may awake immediately, or may continue to sleep.

A condition which is allied to that of hysterical lethargy or trance has long been known as *catalepsy*, a term to which, however, so many meanings have been attached as to give rise to an almost hopeless confusion in literature. In its correct use it signifies a form of morbid sleep which is characterized by loss of consciousness and of voluntary motion, and by a peculiar condition of the muscles, so that the body or the limbs take without resistance any position in which they are placed, and preserve without apparent effort such position for an indefinite time. In some cases of insanity and in other neuroses the muscles pass into a state somewhat resembling that of catalepsy. These are not, however, instances of catalepsy, to which, in truth, the loss of consciousness is as necessary as is the peculiar condition of the muscles. To designate muscular symptoms resembling those of catalepsy occurring in other conditions the term *cataleptoid* may well be used.

Catalepsy generally develops gradually, but it may come on abruptly as the result of a powerful emotion. As an instance of such sudden development may be cited the case recorded in 1415 by Lafaille (*Annales de Toulouse*, 1st part, 1415) of two Gray friars who, during a sermon on the Passion, were struck immovable in the attitude of devotion. Usually the cataleptic status is preceded by lethargy, convulsions, or other pronounced hysterical symptoms. It lasts from two hours to a day, and may

continually recur, so that the patient is said to be cataleptic for many days or even for weeks. The facial expression may be that of apathy; in some cases it is that of devotion, of rage, or of whatever passion the subject was in at the time of the fixation of the muscles. The eyes are wide open, with quiet lids. The body is motionless, in the posture in which it has been placed or in which it has settled during the arrest of active motion. There is no power of voluntary movement, but the limbs are not rigid or contracted. When taken hold of, they bend with the plasticity of wax. In any position in which the body or limbs are placed they remain for a long time, and Berger (quoted by Barth) is said to have seen the most bizarre and difficult attitudes steadily maintained for seven consecutive hours by a young cataleptic woman who was constantly under observation. During the whole of the cataleptic state there is complete anæsthesia of both the common and the special senses, so that the most violent irritations of the skin produce no reaction. Respiration is regular, the pulse maintains its normal rhythm and rate, and the general bodily functions appear to go on unaffected.

A condition resembling that of trance or catalepsy, in which consciousness is preserved, although the patient has no control over the voluntary movements, may develop as the result of a severe acute disease or from other cause. It is affirmed by Barth that in some cases which in other respects entirely resemble those of catalepsy the patient after coming out of the condition has recited all that occurred during the crisis. To those cases of lethargy in which consciousness has been preserved the term *lucid lethargy* has been applied. If in a lucid lethargy the death-like condition spoken of on page 394 has developed, the patient may be thought to be dead. There can be little doubt that under these circumstances premature burial of a conscious person has occurred. In my childhood I well knew an old and esteemed minister of the Society of Friends who, in one of the epidemics of yellow fever, after an attack of the disease, passed into this condition and was prepared for burial. Although perfectly cognizant of his danger, he could give no sign, until by a supreme effort he succeeded in making some slight movement of the eyelids, which indicated life.

If in insanity a cataleptoid condition appears, the true character

of the apparent catalepsy can usually be determined by noticing that the body and limbs when placed in bizarre positions show tremors or other evidences of fatigue. In catalepsy the limb when it falls falls like a wax arm which has been bent and slowly straightens by its own weight.

According to my thinking, it is not at present possible to arrange the various cases of morbid somnolence into symptomatic groups which can be distinguished from one another by the symptoms. In a previous chapter choreic movements were shown to be due to a peculiar condition of the ganglionic nerve-cells, capable of being produced by various causes. It seems to me that it must also be considered that the condition of morbid sleep is due to an altered nutrition of nerve-cells connected with conscious life, which altered condition cannot always be recognized by the microscope, and may be produced by various causes. The parallel between morbid somnolence and chorea is further evinced by the fact that the best classification that we can make of it is etiological. It will at once strike the reader that most of the groups which are separated in the following scheme are represented in chorea:

Group first, those cases in which the unconsciousness is due to a distant reflex irritation.

Group second, those cases in which it is an outcome of a peculiar nervous condition of unknown nature, to which the name of narcolepsy is applied.

Group third, those cases in which it is hysterical.

Group fourth, those cases in which it is connected with insanity.

Group fifth, those cases in which it is due to an organic disease of the brain.

Before taking up the consideration of these etiological groups, it is necessary to call attention to the fact that epileptic attacks may closely simulate morbid sleep.

Epileptic Sleep.—A sleep of some hours' duration out of which the patient cannot readily be awakened is a portion of a typical epileptic attack. In rare cases the whole epileptic paroxysm may be comprised in a sleep which may be prolonged for several days. The following case, reported by Surgeon M. Chabert (*Receuil de Mémoires de Médecine et de Chirurgie militaires*, 1867, vol. xviii.

p. 16), was probably epileptic. A soldier, twenty-six years of age, suddenly disappeared, and was marked as a deserter. Eighteen days later he was found deeply buried in a pile of straw lying in an out-house, the door of which is asserted to have been locked for seven days. The man was completely unconscious and apparently dead. The face was deadly pale, with dilated pupils, the limbs were relaxed, the surface was cold and without sensibility, the respirations were completely suspended, and even on auscultation no evidence of cardiac action could be made out. After half an hour's work with frictions, external heat, etc., an inspiration was taken: in a short time respiration and circulation were re-established, and on the next day the man was conscious. Inquiry showed that at the age of twelve years the patient had been seized with a furious migraine, had disappeared, and was found twenty-four hours afterwards asleep in a granary. Two months later he had a second similar attack. In the twelve or fourteen years after this he had seven or eight attacks, during which he would be deprived of consciousness for several days.

Another case, which may have been hysterical, but possibly was epileptic, is that recorded by Dr. Marduel. The soldier was admitted to the Military Hospital of Lyons on the 21st of March, 1870, profoundly unconscious and insensible, but with a full regular pulse of eighty-four, and calm respiration. In spite of the use of cold affusions and violent electrical currents, the sleep continued for seventy-four hours. Subsequently the patient had another attack of sleeping, during which there was a violent convulsion. The fact that in one of these sleeping periods there was general hyperæsthesia of such character that touching of the skin would immediately produce a violent tetanus or opisthotonos, points towards hysteria.

Reflex Unconsciousness.—It is well known that in poisoning by gastro-intestinal irritants stupor or insensibility may be so pronounced as to mask the ordinary symptoms. I have seen this in a case of poisoning with Paris green. I was once called to a child who was in profound insensibility, with very alarming collapse, apparently without cause, but who, I shortly learned, had eaten stale cream puffs a few hours before. By the use of a hot bath and emetics consciousness was soon restored. The following cases may be cited as instances of reflex unconsciousness.

embolic. The region of the brain involved is supplied by the middle cerebral or Sylvian artery, the line of whose course so corresponds with that of the carotid arteries that the blood-current is very likely to carry into it any foreign matters which may reach the brain. The Sylvian artery on the surface of the island of Reil divides into four branches: of these, the first is distributed to the outer portion of the orbital surface of the hemisphere and the adjacent inferior frontal convolution; the second supplies the chief part of the second ascending convolution; the third passes through the fissure of Rolando to the remainder of the ascending frontal convolution, to the ascending parietal convolution, and to the inferior part of the superior parietal lobule; the fourth branch, lying in the posterior part of the fissure of Sylvius, supplies the inferior parietal lobule and the superior temporo-sphenoidal convolution.

The three speech-centres of Grasset, although conjointly supplied by the middle cerebral artery, are reached by distinct branches of this artery, so that whilst an arterial lesion of the main trunk involves all the speech-centres and gives rise to a complex aphasia, a lesion of one of the branches may involve either of the aphasic regions separately and give rise to one of the special forms of the affection.

In children aphasia sometimes exists without hemiplegia. It may be due to an arrest of development, which, I believe, may be caused by an emotional storm. A case which died under my care had the history of the child's having been well and strong until it was nearly two years old, at which time it was beginning to talk successfully. It was then taken into a railway-train, was excessively frightened, and screamed for two hours. From this time it ceased to talk, developed convulsions, and, after several years, died. At the autopsy the only lesion I could find was complete failure of development of the convolution of the left island of Reil. The brain looked as if this had been abruptly gouged out of it.

GENERAL MEMORY.

Like most functions of the organism, memory may be stimulated, perverted, or depressed.

Exaltation of Memory.—A distinct, indisputable stimulation

or increase of the memory under the influence of pathological processes is a phenomenon rarely to be distinctly recognized. Forbes Winslow details cases in which an extraordinary excitation of the memory and of other mental functions preceded an attack of apoplexy; and it is probable that in the mental exaltation which precedes a general mania or occurs in the peculiarly dangerous form of insomnia due to excitation of the cerebral cortex the memory may share in the general functional excitement of the brain.

Failure of Memory.—Loss of memory is an exceedingly frequent symptom of organic brain-disease. It cannot be said to be characteristic of any particular form of brain-disease, but is liable—indeed, almost certain—to occur in organic affections of the cerebral cortex. Its diagnostic importance comes from the fact that, unless due to obvious acute disease or connected with insanity, it is a strong indication of an organic affection of the brain. A degree of failure may, however, arise from simple brain-exhaustion.

Usually the patient or his friends recognize even a slight loss of memory; but sometimes very careful search is required for its discovery. Under these circumstances the physician must question the patient as to the small events of the last twenty-four hours, and not be misled by that vividness of recollection of the long past which sometimes causes the sufferer to declare that his memory is even stronger than normal. In doubtful cases of general paralysis of the insane, failure of the memory is of special value in enabling us to distinguish the organic insanity from functional mental disturbances which may simulate it. According to my own experience, failure of memory which is not accompanied by paralysis for the time being of all the functions of the mind, as in insanity, is of serious import in proportion to its completeness.

CORRELATED DISORDERS OF MEMORY AND CONSCIOUSNESS.

As has already been stated, a memory is possessed by all varieties of ganglionic nerve-cells, but that intellectual function to which the name is usually restricted is so closely related with consciousness that we can scarcely conceive of its existence without consciousness: nevertheless, the connection of memory with dreaming shows that it is a separate function from consciousness.

There are a good many reasons for believing that the impressions of all events with which an individual has been connected are indelibly recorded upon his brain-tissue, although he may not be able to bring such impressions into conscious perception. At the approach of death, or under the stimulation of disease at a time when consciousness is wanting, persons will frequently speak in foreign tongues, recite passages of prose or poetry long since forgotten, or give detailed accounts of events that occurred in their earliest childhood and of which they have in their normal condition not the slightest remembrance. It would therefore appear that two distinct functions or acts are involved in conscious memory,—one the preservation of the records, the other the dragging out of such records into the light of consciousness and their recognition by the personality of the man. In certain diseases when consciousness is obliterated the connection between the stored records of the cerebral cortex and the automatic speech-centres is so close that the latter act in obedience to the records, and the unconscious patient speaks in an unknown tongue, or relates occurrences of which he has no conscious memory.

When the link that binds consciousness to memory is broken by disease, consciousness may exist without memory. Under these circumstances consciousness is isolated from the past, although the past may still be connected with the present by an automatic unconscious memory.

This is illustrated by the case (Ross, *Nervous Diseases*, vol. ii. p. 880) of a man who was wounded during the Franco-German war in such a way as to lay bare the brain for about two and a half inches in the left parietal region. As the result of this he was subject to attacks lasting from twenty-four to forty-eight hours, in which, although in a condition of apparent partial consciousness, he had no sensitiveness of any part, and was unaware of physical pain. Nevertheless, his will was at once influenced by external objects. If set upon his feet, the contact with the ground started him to walking, when he marched straight on, quite steadily, with fixed eyes and without saying a word. If he met with an obstacle, he would touch it and try to make out what it was, and then get out of its way. A pen placed in his hand started him to writing. Dr. Ross says of him,—

"Give him cigarette-paper and he will take out his tobacco, roll a cigarette, and light it with a match from his own box. But ignite a match yourself and give it him, he will not use it, but let it burn between his fingers. If his tobacco-box be filled with any trash, he will roll his cigarette and smoke without perceiving the hoax. If a pair of gloves be put into his hand, he will put them on, and, being reminded of his profession, will look for his music. If a roll of paper then be given to him, he will assume the attitude of a public performer and begin to sing."

Loss of Personal Identity.—An attribute of the human understanding which is dependent upon the existence of memory and consciousness is the sense of *personal identity,—i.e.*, the conviction of the individual that he is the same person as he has been in the past. The unbroken chain of events recorded from an indefinite past correlated with the consciousness of the present gives the realization of the unity of the present with the past. This sense of personal identity is destroyed by a complete loss of memory, which loss may be abrupt and be unaccompanied by impairment of consciousness or of rationality. I have seen this association of symptoms continue for several days after a sunstroke, so that the patient, who had been brought by ambulance into the hospital, was unable, after he had recovered his mental faculties and was perfectly rational, to give any clue to his personality which could lead to his identification.

Double Personality.—Hasheesh and perhaps some other drugs have the power of producing a sense of *double personality,*—a condition in which the subject feels as though he were two distinct personalities, one holding intercourse continually with the other. In insanity this feeling of double personality may be the basis of delusion. Such delusion usually takes the form of an absolute belief in a dual existence: thus, in a case of my own, an insane man believed that he and all others of the human race had their "doubles," which were not to be distinguished from their proper personalities. The life of the patient was overwhelmed by the constant fear that he was not himself, but his own double.

Double Consciousness.—Double personality is to be distinguished from the extraordinary phenomenon to which the names of *double consciousness, periodic failure of memory,* and *periodic amnesia* have been given. In this state there is undoubtedly a disorder

of memory, but in most cases all the intellectual functions are deeply involved. Before attempting an analysis of double consciousness I shall briefly sketch some of the more important recorded instances. The earliest record of such a case that I have been able to find is by Dr. Mitchell (*Med. Repos.*, p. 185, New York, 1817). A very highly educated young woman fell without warning into a deep sleep, which lasted for many hours. On waking, she had lost all her former knowledge; her memory had become a *tabula rasa*, every trace of her past culture having disappeared. It was necessary for her to relearn everything. After extreme effort she became familiar with surrounding persons and things, acquired the alphabet, then learned to read, then to write, and finally to reckon. Some months later she again fell into a deep sleep, and awoke in her normal state. She now knew all that she had learned in her original condition. For many years after this she alternated between the first and second conditions, in each state knowing only what she had learned in the previous periods of the same state. When she made acquaintances she recognized them again only when she was in the state in which she had been at the time of the first meeting. Her handwriting, which was very good in her first condition, was very bad in her second state.

Dr. Azam (*Annales Méd.-Psych.*, 1876, vol. xvi.) reports a case of double consciousness occurring in an hysterical girl. In this patient the change from one state to the other was always preceded by a profound sleep lasting three or four minutes, this period of repose being ushered in by an intense headache. In her abnormal state the girl was extremely gay and vivacious, and remembered perfectly all that had passed both during previous similar abnormal conditions and during her normal life. There was no delirium, no hallucination, no false appreciation, but the intellectual faculties were more developed than during her normal condition. After this condition had lasted a variable length of time, her gayety suddenly disappeared, her head dropped, and she fell into a deep sleep, out of which she awakened in a condition of great sadness. She forgot all those things that had happened during her abnormal period, but remembered perfectly everything that she had known or that had been done during the preceding normal states. Having been seduced and become preg-

nant during one of her abnormal periods, she was entirely ignorant of the affair during her normal state, although fully aware of it during successive abnormal periods. Finally, while in her normal condition she was made to understand that she was pregnant, when she was seized with violent hysterical convulsions. After the birth of the child she had no mental trouble for several years, and was married. Somewhat later, after a very painful and exhausting accouchement she had hysterical lethargy, followed by ecstasy and violent hysterical manifestations. At thirty-two she was the mother of a family, and an active business woman. The child which had been conceived during an abnormal period was very intelligent, and an excellent musician, but of a highly nervous temperament, and liable to nervous attacks.

A case somewhat similar to this is reported by Dr. James Mayo (*London Medical Gazette*, vol. i., 1845). A young girl passed repeatedly through two alternating different states of mental existence. During the abnormal periods she was extremely excitable, and had mental attributes much above her normal condition. She made progress in needle-work and in intellectual acquirements far beyond what was possible with her natural talent. She also became very vivacious in conversation, but did not recognize her relations to her father and mother, calling them by wrong names. On the subsidence of her abnormal state her recollection of kindred and friends returned, and she resumed her quiet, dull character. In the abnormal state she remembered without the slightest confusion all that had happened in previous abnormal periods, and what she had learned either manually or intellectually, but knew nothing of what had occurred in her normal conditions. In her normal conditions she had no knowledge of anything that had happened or of anything that had been learned during the abnormal states.

I have seen one case which offered symptoms resembling those of double consciousness. They were produced by a depressed fracture caused by a blow upon the head. Previous to trephining there were at least four of the abnormal states, but since the operation, over a year ago, there has been no return, although the patient's mental condition is not good. In the first of his spells he went to a railroad-dépôt, bought a ticket, travelled on the cars two or three hours, and, after getting out, met an acquaintance,

who expressed great surprise at seeing him, and asked why he had come, receiving the reply that he had come to try to get work. The man then went to a friend's house to dinner, where he talked and ate, until suddenly he waked up, with an inquiry of intense surprise as to how he had got there. He appeared to have no recollection of his trip, except of a few minutes, his remembrance of which was so vivid as to indicate that he had then had a waking spell. During the abnormal period the man's behavior suggested to his acquaintances only that he was distraught. I was unable to obtain evidence that he exhibited during the spells any memory of acts performed in previous similar periods.

It does not seem necessary to abstract any more of the few recorded cases of double consciousness for the purposes of present illustration. In a typical case there is, first, an abrupt loss of memory at the beginning of each paroxysm for everything that has happened during paroxysms not of the same series; secondly, a change in the personal character of the individual, the disposition, the habits of thought, and even the intellectual powers being altered.

Double consciousness may exist in various degrees. Thus, in a case reported by Dr. Samuel Jackson (*Amer. Jour. Med. Sci.*, 1869, p. 18), the character was affected rather than the memory. An hysterical young lady was attacked with nausea and vomiting, followed by a complete alteration of character and change of voice. Formerly mild and gentle, she became abrupt and rude, and yet would so perform her household duties that it was often difficult to determine in which condition she was. The attacks came on daily, without apparent cause. The symptoms in this case seem to represent only an exaggerated form of moodiness; and there can be no doubt that even the most complete double consciousness is closely related on the one hand to epilepsy and on the other to hysteria and to insanity. In my case detailed above, the cause of the attack was a blow upon the head,—a not rare cause of epilepsy,—and there was no binding memory between the spells. In that form of epilepsy in which there are automatic movements, and also in cases of epileptic delirium, the paroxysm is in many ways related to the second period of double consciousness, but differs in that the individual does not preserve any reasonableness or capability of being affected by other persons,

and does not remember what has occurred during previous spells of similar type. On the other hand, in a large proportion of the cases, double consciousness has occurred in hysterical women, and the phenomena pass almost insensibly into those of hysterical semi-consciousness and delirium.

In insanity there is often a change in the whole manner of thought and character of the individual. In a case of melancholia long under my care, the patient after recovery was subject to frequent attacks of transitory melancholia, which lasted from a few to many hours: whilst perfectly contented and happy, she would say, "It is coming," and in a few moments would be covered over as it were with a wave of emotional depression which would, for the time being, completely alter her habits of thought and her behavior. In a case of profound apathetic melancholia recently at Burn Brae Asylum, near this city, the patient one day suddenly said, "I have had a revelation: my sins are forgiven me," and, after weeks of absolute voicelessness, became talkative, rational, and active in all his sympathies, without, however, being unduly excited. Not long after this the man relapsed into his insane condition. It is said that similar sudden changes have previously occurred several times. This man appears to carry his memory over from one state to the other, and in this only do his symptoms differ from those of typical double consciousness. If in such a case of insanity as this the continuity of memory should be broken, there would be a typical double consciousness.

The close relation between insanity and double consciousness is further illustrated by a case reported by Dr. David Skae, in which a man after eighteen months of typical melancholia developed a twofold life, being on alternate days sane and insane. On melancholic days he neither eats, sleeps, nor walks, but sits incessantly turning the leaves of the Bible and complaining piteously of his misery. At this time he has no remembrance of the days in which he is well, nor of any engagements made during them: he does not, and cannot be made to, recognize the existence of such days, but contemplates the future with hopeless despondency. On the alternate well day he denies that he has any cause of complaint, believes that he was well the previous day, transacts business, takes food and exercise, and is entirely free from delusions or despondency. He also anticipates no

return of his illness, and has no memory of his bad days. He remembers exactly the transactions of his previous well days, and persists in making business engagements for the following day,—*i.e.*, for his melancholic day,—although repeatedly assured that at the time named he will be unable to attend to business.

There is a very rare mental condition known by German writers as *Doppeltwahrnehmung*, or *double perception*, which is liable to be confounded with the condition I have just described under the name of double consciousness. The peculiarities of this affection are well portrayed in the report by Dr. M. Ruppert (*Allgemeine Zeitschrift für Psychiatrie*, 1869, vol. xxvi. p. 531) of a case in which whenever the man read to himself he would plainly hear each word repeated as though a chorus of fifty or sixty female voices were speaking to him, and when he ceased to read he would hear the last words read after him. This reading after him disappeared as soon as he spoke aloud, and was prevented by his reading aloud. In one sense of the term consciousness, this patient had a double consciousness; but it is plain that his symptoms were much more closely related to hallucinations than to the state commonly known as double consciousness.

CHAPTER X.

DISORDERS OF CONSCIOUSNESS.

In a book like the present an elaborate discussion of the so-called physiological theories of sleep would be out of place. It seems to me, however, that a few words upon the subject are required. According to some physiologists and neurologists, natural sleep is induced by the withdrawal of blood from the brain. It must be allowed that the concordant results of experiments show that during sleep there is a more or less pronounced cerebral anæmia, which on awaking is replaced by turgescence of the cerebral vessels. This is not, however, proof that sleep is induced by the withdrawal of the blood. It is a universal law that cessation of functional activity is immediately followed by lessening in the amount of blood in the part. I conceive, therefore, that the sleep is the cause of the bloodlessness, and not the bloodlessness the cause of the sleep. This is certainly in accord with clinical experience, which to my mind proves that insomnia may be connected either with excessive anæmia or with excessive congestion of the cerebral cortex. Thus, the wakefulness of anæmia is well known, as is also the insomnia of acute mania. Some physiologists have attempted to explain the production of sleep by supposing that certain chemical compounds are formed during the activity of the day, which, circulating in the blood of the brain, act as hypnotics on the cerebral cells. There is no evidence worthy of attention establishing any theory of this character, and to my thinking such chemical theories are upon their face so improbable that they should be received only after the clearest proof. Concerning sleep, as concerning other functions of the human organism, the simplest explanation is the most probable. In the greater portion of the active tissues of the organism rest alternates with activity, and the brain in its sleep conforms to this general habit. The best explanation of sleep, then, is that when exhausted by effort the cortical brain-cells pass into a condition of functional inactivity, during which their power of further effort is

recuperated. Because consciousness is the expression of functional activity in these cells, therefore when these cells do not exercise their function there is unconsciousness,—*i.e.*, sleep. That sleep, or functional rest, should be more or less periodical seems essential from the very nature of the case. Throughout a healthy nervous system a tendency to periodicity of action is marked. In disease this tendency becomes even more apparent. Pain-storms recur with more or less regularity, habits of periodical discharge, at first accidental, become fixed, convulsions develop at intervals, etc. The daily rhythm in the production and dissipation of animal heat during health is a forerunner of the marked diurnal swing of temperature so common in fevers.

The bearing upon the practice of medicine of our belief as to the immediate causation of sleep is very apparent. If we think that insomnia is due to cerebral anæmia, we must treat the anæmia to remove the insomnia. Out of such error have grown other theories, which, though absurd, have been largely dominant. A notable example of these is the belief, at one time wide-spread, that bromide of potassium produces sleep by affecting the blood-vessels. Again, largely in order to sustain their theories, certain neurologists have distinguished sleep, stupor, and coma as essentially diverse conditions readily to be diagnosed in the sick-room. These states are, however, simply the outcome of different degrees of completeness in the suspension of the functions of the cerebral cortex: such a suspension finds its lightest expression in a doze, and its profoundest development in a coma. No rules of diagnosis can be laid down which will enable us to draw any practical lines, sharp and fixed, between the lightest slumber and the most complete unconsciousness. Nor is the unconsciousness of anæsthesia an isolated thing. It is simply a suspension of cerebral function in which a chemical agent is the cause of the paralysis. In the sick-room every grade can be found between light and heavy slumber, between heavy sleep and stupor, and between stupor and coma. For the purposes of discussion we must, however, arbitrarily separate these states. I would define these terms as follows: *sleep* is that condition of unconsciousness in which the subject is readily aroused, and when aroused is easily kept awake by ordinary external stimulations or by his will-power; *stupor* is that condition in which the subject is aroused with great

difficulty, and when left to himself relapses into unconsciousness; *coma* is that state in which it is impossible by external irritation to restore consciousness.

In the present chapter I propose to treat of, first, sudden loss of consciousness; secondly, sleep, its abnormalities and accidents.

SUDDEN LOSS OF CONSCIOUSNESS.

Sudden loss of consciousness is a symptom of such varying import that it is scarcely susceptible of scientific arrangement in a treatise like the present. Nevertheless, it is one of such practical importance as to demand discussion. A blow upon the head, or even upon a distant part of the body, may produce immediate insensibility; but the study of such cases belongs to surgery, and I shall in the present consideration of the causes and collateral symptoms of sudden unconsciousness omit traumatism and its results. Loss of consciousness is an essential part of the epileptic convulsion; but, as this convulsion has already been studied in detail, I shall at present consider only affections in which there are no convulsive symptoms, or in which if the convulsion occurs it is not an essential feature of the disease, but an incident of the attack. Thus, in epilepsy the convulsion is an essential feature, but in a cerebral hemorrhage or a sunstroke it is not of such character; and if a convulsion occur in an apoplexy it belongs to the individual case, because it is not necessarily present in the disease.

The ordinary non-traumatic causes of the sudden loss of consciousness are epilepsy, hysteria, syncope, sunstroke in its various forms, apoplexy, various forms of poisoning, and malignant systemic diseases.

The symptoms which attend the *hysterical* and the *epileptic* loss of consciousness, and the methods of recognizing the nature of such attacks, have been thoroughly discussed in a previous chapter. *Syncopal* loss of consciousness, due to a failure in the supply of blood to the brain, is to be recognized by the extreme pallor of the subject, by the absence of the pulse, or its excessive threadiness or weakness, at the wrists, and by the greatly enfeebled action of the heart, as shown by the weakness of the cardiac sounds and impulse. Its diagnosis is facilitated by noting that it occurs after

exertion or during great excitement, and in a subject already enfeebled by disease, hemorrhage, or accident, or else of a naturally weak constitution.

Sunstroke.—During the summer months sunstroke is a very frequent cause of sudden loss of consciousness. It develops only after exposure to heat, either natural or artificial. It may happen in the day or in the night, and is especially fatal in sugar-refineries and other places where the heated air is saturated with moisture. It occurs most frequently in unacclimated races, and is very rare in negroes, Hindoos, and other tropical peoples. There are two distinct forms of it,—one of which may be known as thermic fever, the other as heat-exhaustion.

The animal organism is constructed to run upon a certain plane of heat, and whenever this level is departed from all the functions of the body suffer. The nervous system is the most susceptible portion of the organism, and therefore the nervous symptoms are always prominent when there is a great disturbance of the bodily temperature. It appears to make little difference whether the temperature be elevated above or depressed below the norm, so far as the nervous system is concerned. In either case, if the departure from the norm be sufficient, consciousness is lost, the lower brain-functions are implicated, so that the respiratory and cardiac action both become irregular, and death occurs at last usually from paralysis of the respiratory functions. In thermic fever the temperature of the body is greatly elevated. In heat-exhaustion the bodily temperature is depressed.

Heat-exhaustion is very often felt in a mild degree by feeble workers in hot weather. There is a sense of weariness and distress, pallor of the countenance, failure of the muscular force, and finally failure of the pulse. Severe examples of the affection are rare. In these the symptoms may develop almost as suddenly as in thermic fever. Thus, in a case brought into the Centennial Hospital during my service in 1876, there was sudden unconsciousness, with muttering delirium; great restlessness; a facial expression of collapse; profuse perspiration bedewing the whole surface; rapid, feeble, scarcely perceptible pulse; and a mouth-temperature of 95°. Although in their general aspect the symptoms of heat-exhaustion resemble those of thermic fever, the true character of the case should be at

once recognized on touching the ice-cold surface of the body. The only disease or condition readily confounded with heat-exhaustion is collapse from other causes. I have seen cases of internal aneurism, of pernicious malarial fever, and of other affections picked up in the streets and brought into the hospital in collapse in which a mistake in diagnosis would have been very excusable. If such a case should happen upon an intensely hot day, and the bodily temperature be much below the norm, the diagnosis might have to be reserved. Except as regards pernicious malaria, however, this is a matter of little importance, because the treatment of heat-exhaustion is the same as that of collapse with lowered temperature from other causes.

Thermic fever, *heat fever*, or *coup de soleil* (sunstroke) usually comes on without distinct prodromes, although frequently there is a great sense of distress or of a general burning heat before the loss of consciousness, which may also be immediately ushered in by chromatopsia, or colored vision,—the whole landscape being deluged in a blue, yellow, or red light. The unconsciousness ordinarily develops abruptly, and is complete, although very frequently it is associated with muttering delirium. There is usually great muscular restlessness, which in some cases becomes convulsive or is replaced by violent epileptiform convulsions. Sometimes the patient is profoundly relaxed and quiet. The surface of the body, at first dry, often later in the attack gathers upon itself an excessive perspiration, which does not, however, reduce its burning heat. The face is flushed, and the eyes are suffused. The rapid pulse is sometimes bounding and apparently strong, although almost invariably compressible; frequently it is feeble and even thready, especially if the symptoms have lasted for some hours. Vomiting is very common; purging is in bad cases almost always present. The whole body is apt to exude a peculiar odor, which is especially strong in the fæcal discharges. The characteristic symptom is the high temperature, which, as measured in the mouth or rectum, may reach 112° or 113°, and is rarely below 108° in cases severe enough for unconsciousness to be present. The urine is scanty, sometimes albuminous, not rarely finally suppressed. The breathing is more or less labored, and often irregular, and towards the last generally becomes more and more shallow. Although at times the patient suffering from

thermic fever may be partially aroused by shouting, shaking, etc., the unconsciousness is often absolute. The pupils are variable, sometimes contracted, sometimes dilated.

The diagnosis of this form of thermic fever is usually free from difficulty: the known exposure to heat,—*i.e.*, to the cause of sunstroke,—the unconsciousness, and other symptoms, and the very high temperature both of the surface and of the interior of the body, are characteristic. If, however, an apoplexy should occur upon a very hot day and be, as it might, associated with a sudden rise of temperature, the diagnosis would not be easy; indeed, if the mouth were not drawn and the general relaxation prevented the recognition of hemiplegia, the diagnosis might be impossible.

In this country the profession has been accustomed to recognize as sunstroke, or thermic fever, only the severe cases which approach to the symptoms that have just been detailed. For many decades, however, medical practitioners in India have known that there is a form of continued fever due to heat which, under treatment, may gradually subside, or which may at any time end in a sudden explosion like that of true sunstroke. Dr. John Guitéras (*Therapeutic Gazette*, March, 1885) has shown that this mild form of thermic fever occurs in the subtropical portions of the United States. These cases have usually been supposed to be instances of typhoid fever, from which, according to Dr. Guitéras, they are to be separated by the suddenness of their onset, the temperature of the first day reaching 103° F., or even higher; by the course of the fever, which is extremely irregular, indefinite as to duration, and almost always has the morning remission more accentuated than in typhoid fever; by the absence of petechia, miliaria, rose-colored spots, tenderness, and gurgling in the iliac fossa; by the tongue remaining moist, with only a light creamy coating, interrupted by a clear streak along the edges and median line; by the absence of cerebral symptoms, except at times when the fever rises very high; and by the mental alertness instead of hebetude. Diarrhœa, although often absent, may be present, and even be severe and bloody.

Many cases of obscure indisposition during intense hot weather, especially in children, are really mild instances of derangement of function of the body by heat,—that is, of thermic fever. Dr. C. Comegys, of Cincinnati, was the first to call attention to

the fact that cases of *cholera infantum*, so called, are frequently instances of thermic fever and yield at once to the use of the cold bath. It has long been known that in cholera infantum there is often a sudden increase in the fever, with or without lessening in the number of the passages, but with a rapid loss of consciousness, which is likely to end in death. Under these circumstances the cause of the cerebral symptoms is the elevation of the temperature. As it occurs in our large cities in hot weather, cholera infantum is frequently, if not usually, a form of thermic fever, and yields with great readiness to the systematic use of the cold bath. In all cases of this character the physician should use a thermometer, and if the temperature be found distinctly above the norm it should be reduced by systematic cold bathing. (See article on "Sunstroke," *Encyclopædia of Medicine*, vol. v., Philadelphia, 1886.)

Apoplexy.—The term apoplexy, as used in this work, applies to cases in which consciousness is suddenly lost as the result of disturbance in the circulation of the brain. Scientific accuracy would require that cases of arrest of the cerebral circulation should be separated from cases of congestion or hemorrhage: the diagnosis, however, between loss of consciousness which is the result of arrest of circulation in the brain by an embolus and the unconsciousness which is due to a sudden rupture of a vessel and hemorrhage into the brain is frequently not possible. Moreover, it is by no means certain that even in hemorrhagic apoplexy the suspension of cerebral functions is the result of a congestion or of an excess of blood in the brain. For these reasons it seems to me wisest to consider under one heading all losses of consciousness connected with circulatory brain-disturbance, and afterwards to point out the few facts of value that we have as guides in separating between the forms. Again, it should be said that, except by the presence of paralysis or convulsions, we have no way of distinguishing between an apoplexy which is simply due to congestion of the brain and one which is connected with more or less permanent organic changes. It has been denied that there is such a thing as simple *congestive apoplexy;* but there can be no doubt of its occurrence. I have seen it developed without obvious cause, to be recovered from without sequelæ, and never to recur in after-life. A congestive apoplexy may be due to a brain-tumor, or to a

syphilitic disease of the brain or its membranes. For a detailed discussion of these comas, see article on Organic Coma.

For description and study, cases of apoplexy may be arranged in two classes, to which the names of sthenic, or congestive, and asthenic, or syncopal, may be given.

There are no etiological differences between these two classes. Extreme typical cases differ widely and characteristically in their symptoms, although they rest upon similar structural changes. Moreover, every possible intermediate variation occurs in nature between an extremely sthenic and a typically asthenic apoplexy, so that the two classes are not naturally separated from each other, but are artificially characterized for the purposes of discussion.

The apoplectic attack may come on with great suddenness. It may, however, be preceded by prodromes, which are in some cases affirmed to have lasted for some hours. Sometimes without distinct warning the patient drops unconscious, or he may become confused in speech and manner, and then suddenly be stricken, or else gradually grow more and more heavy and finally sink into unconsciousness. At the height of the attack the unconsciousness is complete. The pupil is fixed, dilated or contracted, as the case may be. In the congestive form the face and conjunctivæ are intensely suffused, dark purplish red. The breathing is loud, snoring, and stertorous. The pulse is usually full and bounding. It may be slower or more rapid than normal; occasionally it is small and hard. The surface is warm.

In the syncopal form the face is pale, and the breathing quiet, or, if stertorous, not loud and harsh in its sound. The surface is coolish, and the pulse rapid and feeble.

Paralysis, convulsions, and great rise of temperature may occur in either variety of apoplexy. They are proof of the presence of an organic lesion. The apoplexy may end in death, which is commonly developed by a more or less rapid increase of the symptoms. The unconsciousness remains complete; the pulse, whether originally strong or feeble, continually falls in force; the respiration grows more and more shallow, or more and more irregular, and may at last gradually die away or suffer sudden arrest. The cerebral reflexes are frequently lost early in a case of severe apoplexy, especially when the basal region is invaded by the lesion. Thus it is that the power of swallowing is affected. Complete

loss of this function is a very serious if not a fatal symptom. When recovery occurs it is usually gradual, although in rare cases the patient may arouse himself suddenly. Even when the patient seems rational, mental action may still be very imperfect.

Unconsciousness from Indirect Causes.—Unconsciousness more or less closely resembling that of apoplexy occurs from a multitude of causes. Such unconsciousness is usually developed gradually, and is accompanied by other symptoms which at once distinguish it from the unconsciousness of apoplexy. With a clear history there is rarely any difficulty in distinguishing these various forms of unconsciousness from that due to disturbances of the cerebral circulation. Very frequently, however, in hospital practice the physician is called upon to make immediately such diagnosis as may be necessary for treatment without having knowledge of the antecedents of the attack. The importance and difficulties of such diagnosis seem to me to justify the consideration in this place of the more important forms of unconsciousness which are likely to be brought into a hospital.

In the first place, the patient should always be carefully examined for the evidences of traumatism,—cuts about the head, bruises, etc. I once saw at an autopsy upon a man who had been brought into the hospital unconscious, and in whose case there was no suspicion of traumatism, a knife-blade projecting two inches into the brain, and broken off close to the outside of the skull. A traumatism may be present without the physician being able to detect it, but usually some local indications of the injury will be revealed by close inspection. In the case just spoken of the wound was very narrow and small, and was entirely concealed by the bushy hair.

Loss of consciousness may be caused by poisoning and by great emotional disturbance, or it may be an early symptom of acute blood-diseases. Unconsciousness from acute alcoholism is very frequent in large cities. Not very rarely persons supposed to be only dead-drunk are put into station-house cells, to be found dead some hours afterwards. Even when suspicion is aroused, the diagnosis between apoplexy and deep intoxication may be very difficult. The odor of alcohol about the breath or the person, and its presence in the urine, point towards alcoholic poisoning, but cerebral hemorrhage may occur after either moderate or im-

moderate drinking. It is in such cases that mistakes are most liable to occur. Whenever by shaking, shouting in the ear, etc., a drunken person cannot be aroused, the probabilities of cerebral hemorrhage are strong enough to justify the temporary diagnosis and the institution of proper measures of relief. A very careful examination should be made for evidences of paralysis. Under the conjoint influence of alcohol and cerebral hemorrhage the universal muscular relaxation is so complete that the detection of a local palsy may be a matter of the greatest difficulty. Drawing of the face to one side is of course decisive, and if in breathing the air comes out with a sort of puff and pulling of one corner of the mouth, the case is one of cerebral hemorrhage. If the patient be at all restless, the motionlessness of the arm or leg of one side will usually betray a hemiplegia. If the pupils are unequal, the probabilities are in favor of cerebral hemorrhage. In any doubtful case the physician should be very careful not to affirm that the patient is suffering simply from drunkenness, but should reserve his opinion.

Opium-poisoning produces symptoms very similar to those of apoplexy, and I have seen cases in which the diagnosis was simply impossible. The presence of hemiplegic or monoplegic palsy is, of course, decisive. But I have seen inequality of the pupils produced by opium. The remarks made in regard to distinguishing alcoholic unconsciousness apply with equal force to opium-poisoning in its advanced stage.

Uræmic poisoning is a very frequent cause of unconsciousness, and, unlike alcoholic and opium poisoning, is often associated with convulsions. The diagnosis between uræmic and organic convulsions has been already discussed. (See page 118.) It is sometimes impossible to make an immediate diagnosis between the quiet form of uræmia and cerebral hemorrhage, especially since in uræmia serous effusion into the cerebral ventricles is common. Cerebral hemorrhage itself is not rare in advanced kidney-disease. In every case brought into a hospital unconscious the urine should be at once examined. If it be normal in specific gravity and free from albumen, the probabilities are strongly against uræmia. If the urine be scanty and of low specific gravity, much more if it be albuminous, the patient is probably suffering from kidney-disease. The presence of local palsy, even though there

be advanced renal degeneration, is demonstrative of either hemorrhagic or serous exudation into the brain. A temperature above the normal without convulsions would, according to the teaching of the French clinical school, prove that the patient is suffering from something else than uræmia. I, however, doubt very much the correctness of this teaching. (See page 121.) Usually, but not always, the patient in simple uræmia can be aroused to some slight extent.

A partial unconsciousness may result from various *irritative poisons* which do not commonly affect the cerebrum. I have seen a marked stupor amounting to almost complete unconsciousness the chief symptom of poisoning by Paris green, and there are numerous cases on record in which the ingestion of large doses of tartar emetic or other violent irritant has been followed by symptoms similar to those of my case. In some of these cases when no history is obtainable a correct diagnosis may be almost impossible: nevertheless a trifling circumstance, if the physician be sufficiently alert, may give the necessary clue. The circumstances of the case, the friends of the patient, and, if he be at all sensible, the patient himself, should be most carefully examined.

A very important class of cases is those in which collapse with more or less complete unconsciousness occurs as an early symptom of severe *internal disease*,—either a local affection, such as a gastric or an intestinal perforation, the rupture of an aneurism, etc., or a general blood-disease.

With a clear history the diagnosis may be very easy; but often in practical life the history is misleading or altogether absent. The suspicion of the physician should be aroused by noticing that the loss of consciousness is not complete and is accompanied by evidences of cardiac failure, coolness of the skin, and a peculiar "decomposed" expression of the countenance seen only in mortal illness, and usually known as the Hippocratic countenance. It is impossible to describe exactly this facial expression, but to the experienced eye it is sufficient for the recognition of the gravity of the disease. In any case offering the symptoms just described, very careful examination should be made to detect the existence of a local lesion, and if evidence be found of a perforation or of any sufficiently severe local affection, the diagnosis becomes clear.

When collapse, with more or less incomplete unconsciousness, is

an early symptom of a malignant systemic disease, we are forced to make the diagnosis by the process of exclusion. Usually it can be determined that the attack is due to some blood-disease, although often it is impossible to know the exact nature of such affection. Except in the case of a pernicious malarial paroxysm, an accurate diagnosis is not of great importance, because it has little bearing upon the treatment. It is, however, vital to recognize the true nature of a malignant chill. This can usually be done by excluding other causes of collapse and by paying attention to the following positive indications:

First. The unconsciousness is partial: the patient can be aroused, and when aroused speaks in a feeble, usually whispering, voice.

Secondly. There are evidences of internal congestions, such as almost complete absence of respiratory murmur, vomiting, gastric or abdominal tenderness, increased percussion-dulness over the liver or spleen, suppression of urine, etc.

Thirdly. The temperature is disturbed: it may be distinctly lowered; or in some cases there is a low external temperature with a high internal temperature.

Fourthly. A history of previous malarial attacks or of exposure to malarial influences can be obtained, and the season of the year is usually that at which malarial fevers prevail.

Fifthly. The patient is an adult, and does not present paralytic or other symptoms characteristic of cerebral hemorrhage. In children malignant malarial paroxysms do not occur, at least in this climate (that of Pennsylvania), whilst primary collapse from acute malignant constitutional affections, not malarial, is in adults excessively rare.

In any doubtful case the patient should be given the benefit of the doubt, and when the collapse has been recovered from, fifty grains of the sulphate of quinine should be administered during the next twelve to eighteen hours.

Collapse with more or less complete loss of consciousness may result from *emotional excitement.* Many years ago, during a cholera epidemic, in answer to a sudden professional call, I found a man in collapse and partially unconscious, who, when aroused, said in a suppressed, whispering voice that he had had a sudden furious attack of vomiting and purging, which had ceased but had left him in his present condition. I supposed that the case was

one of cholera; but the sequel proved that shortly before I saw him the man had cut the throat of a prostitute after cohabitation with her and left her dead in her bed. The collapse I witnessed was caused by the emotional reaction which developed when the madness of his orgy had passed off sufficiently to allow him to recognize the results of his actions.

SLEEP—ITS DISORDERS AND ACCIDENTS.

In treating of sleep and its disorders I shall divide the subject into three parts:

First. Abnormal wakefulness.

Second. Abnormal somnolence. Morbid sleep.

Third. Accidents or groups of symptoms which occur during sleep, and which are not elsewhere spoken of in this book.

ABNORMAL WAKEFULNESS.

In cases of *simple insomnia* the form of the sleeplessness varies. In some instances the subject is simply unable, when bedtime comes, to go to sleep. In other cases he goes to sleep readily, but in the course of two or three hours wakes, and is unable to slumber again. The latter form of insomnia, in my experience, is not commonly the precursor of severe mental affection. This form of insomnia is, on the other hand, often obstinate.

Insomnia may be prodromic of various diseases of the brain. It is very common in the insanities. It is also present not rarely in such general organic brain-diseases as general paralysis of the insane, but is seldom a symptom of tumor or other focal brain-lesion. It may be produced by various diseases of organs other than the cerebrum. It may exist, however, in its most aggravated form without other evidences of cerebral disturbance, and in some cases cerebral exhaustion, and even more severe mental symptoms, are without doubt produced by the loss of sleep. The diagnosis of the cause of an insomnia is to be made by exclusion. If other symptoms of cerebral disease are wanting, the condition of the heart and kidneys should be carefully examined, because latent disease of these organs occasionally has sleeplessness for its chief manifestation. When no disease of the brain or other portions

of the organism can be made out, the diagnosis of simple or functional insomnia must be settled upon.

MORBID SLEEP.

In studying the phenomena of disordered or abnormal sleep, I shall first consider those disarrangements of the function which occur in acute fevers or other diseases not directly connected with the brain.

Disorders of Sleep connected with Acute Fevers.

It does not seem necessary to occupy space in the present volume with any elaborate description of the various derangements of sleep which occur in acute fevers. Such derangements may consist of morbid wakefulness, or of a peculiar wakefulness with delirium and partial unconsciousness, or of true stupor or coma. In some cases these manifestations depend upon high bodily temperature; they may also be produced by an excessively low bodily temperature. In other cases of fever the disturbances of sleep are probably the result of impaired brain-nutrition, due to the alteration of the blood. An improper aeration of the vital fluid, such as occurs in severe pneumonia, may for a time cause aggravated wakefulness, but more usually it manifests itself in stupor and coma.

In some cases of fever the patient will awaken from a sleep of several hours' duration, and insist that he has never closed his eyes during the time, probably because his dreams have been so vivid that they have conveyed the feeling of wakefulness. To this state the name of *coma-vigil* was given by Chomel in 1834. In 1849 the term was applied by Dr. Jenner to a condition entirely different from that just spoken of. In the coma-vigil of Dr. Jenner the patient lies with his eyes wide open, gazing into vacuity, his mouth partially opened, and his face pale and devoid of expression. The pulse is rapid and thready or imperceptible; the movements of respiration can scarcely be made out; whilst the cold moist skin marks the presence of a deadly collapse. The patient is awake, but is absolutely indifferent to everything that is going on about him. In some cases this state is preceded by somnolence. It is much more frequent in typhus than in typhoid fever, and, according to Murchison, in typhus fever

it is an invariably fatal symptom. I have seen it in typhoid fever in a single case followed by recovery.

Nelavan.—Nelavan, or the so-called *African sleeping disease* (*hypnosis* of Drs. Daguaire and Nicolas), is an acute fever in which excessive somnolence is the most characteristic symptom, and is probably dependent upon the direct action of the poison in the blood upon the brain-cortex. First described in 1819 by Dr. Winterbottom, this affection has of recent years had numerous expositors. It is endemic on the west coast of Africa, but appears to occur epidemically in some of the West India islands. It attacks the negroes especially, but has in a number of instances decimated regiments of French troops. In most cases it comes on gradually, but it may begin brusquely. There is at first a slight frontal headache, with a sense of constriction in the forehead, attended by a mild fever. The vision may at this period be disordered. The gait becomes irregular, and not very infrequently there is a distinct ataxia. Even during the first hours of the headache an intense desire for sleep is manifested. This continually increases until the patient is overpowered by an irresistible somnolence. During the period of sleepiness the strength fails, the spirits are depressed, and there is some fever, but usually neither diarrhœa nor constipation develops, and the forces of the circulation are well maintained. The somnolence when once developed continues to become more and more intense, and the patient gradually sinks into a profound coma, out of which he cannot be aroused. There is at this time complete loss of sensibility. The coma may pass quietly into death; but frequently there are more or less violent convulsions, and in some cases sloughing bed-sores mark the failure of nerve-power. There are no pathognomonic post-mortem lesions, but in 1873 Dr. Macarthy called attention to swelling of the glands in nelavan as characteristic. In this he is confirmed by Dr. Nicolas (*Rev. Méd. de la France et de l'Etranger*, 1880). The disease is very fatal: out of one hundred and forty-eight cases seen by Guérin at Martinique but one recovered (*Thèse*, Paris, 1869, No. 201).

Abnormal Sleep.

The discussion of morbid somnolence is in the present state of our knowledge attended by much difficulty, as it seems impos-

sible to draw the lines between the different varieties of unconsciousness, and it is not always possible to give a clear explanation of the cause of the symptoms. In its simplest form the symptom consists of an excessive drowsiness, which causes the subject to fall asleep at any time during the twenty-four hours, even when in the midst of work or conversation, and leads to his passing many hours daily in bed. When the morbid tendency is a little stronger, the impulse to sleep becomes irresistible, and its influence is so continual that the waking periods may be reduced to only a few hours out of a week. No distinction can be made between the sleep that occurs in many of these cases and that of normal slumber. The cases grade regularly from the person who is simply known as a profound sleeper, to the subject who passes most of his time in unconsciousness. In lighter cases the patient can be awakened out of the sleep, but in the more severe forms of the disorder it is not possible to arouse the sleeper. Here also a regular series of cases exists between the sleep out of which the subject is easily aroused and that out of which he cannot be awakened at all.

In the more prolonged cases of excessive sleep the patient remains in a condition of insensibility for weeks, months, or years. Dr. Guéneau de Mussy reported to the French Academy of Medicine the case of a woman who was said to have gone to sleep in 1808, and to have been still sleeping at the time of the report, January, 1838. During this time the woman remained immovable upon her bed, with her limbs drawn up over her stomach. At first she took food, but soon she ceased to receive any nourishment except the sacrament, which was administered on the first Sunday of every month. As this case appears to rest solely upon the authority of a French curé, and not to have been seen by the doctor himself, the account must be accepted with allowance. There are, however, similar instances in literature. Thus, a case is recorded in the *New York Medical Gazette*, iv., 1853, in which a man slept five years; one in the *Richmond Medical Journal*, 1867, in which a girl slept eighteen years; and the "sleeping girl" of Turville is affirmed to have not awakened for ten years (*Lancet*, June, 1880). Dr. Gaulke (*Vierteljahrsch. für Gerichtliche Medicin*, vol. xxvi., 1877) gives an account of a prisoner who for two years had been in a death-like stupor. Dr.

Blondet details the history of a young woman who slept twelve months. Although the correctness of some of these reports is open to doubt, other cases might be cited, and it cannot be gainsaid that the unconsciousness may last for years, either unbroken or interrupted by brief intervals of consciousness.

Morbid somnolence of a mild type passes by insensible gradations into that condition known by some English authors as *trance*, and usually spoken of by French writers as *lethargy*. Trance, or lethargy, may or may not be ushered in by marked hysterical symptoms, such as immoderate depression or excessive gayety, convulsions, hallucinations, etc. Usually in the course of a very short time the symptoms are fully developed, and the subject is in absolute repose. The face may be red and hot, especially in the first days of the attack, but usually it is pale. The pulse at first may be regular and slow, but after a long sleep it is rapid and feeble; the respirations, generally quiet, may at times become hurried, irregular, and even stertorous. In severe cases the movements of the thorax may be so slight as to be traceable with difficulty. The muscular system, often thoroughly relaxed, may be rigid, and in many cases muscular relaxation alternates with muscular contractions, or even contractures. The eyes are opened or closed; very frequently minute tremors affect both the lids and the eyeballs. The jaws are often set, and sometimes an excess of saliva or even foam gathers about the mouth. In the profoundest cases there is complete anæsthesia of both the common and the special senses, so that neither pinching nor cutting, neither cold nor heat applied to the skin, elicits response. The pupils are usually dilated; they often respond to a powerful light, which, however, calls forth no other signs of life. Sometimes the patient can be readily fed by means of a spoon, but generally in severe cases it is necessary to use the œsophageal tube. Usually digestion is good, but the stools are at long intervals and scanty. The urine is in most cases scantily secreted, and is passed involuntarily. Considering the small amount of nourishment taken, the bodily nutrition is often surprisingly maintained, but in prolonged cases there comes, sooner or later, great emaciation. The bodily temperature may in the earlier parts of the attack be somewhat elevated, but ordinarily it is distinctly subnormal. The awaking is usually, but not always, sudden. During the

course of such a lethargy the subject may pass into a condition which has been mistaken for death. The bodily temperature falls, the respiration becomes so passive that no movement of the thorax or abdomen is perceptible, and, unless a feather or other light object be held to the mouth, breathing may seem to have ceased. The beats of the heart diminish in frequency and in force, so that they become imperceptible even upon auscultation. The face takes on the waxy whiteness of a corpse. The muscular system is in complete resolution, the dilated pupil no longer reacts to light, and even the cornea is filmy as in a corpse. This death-like condition may last for only a few hours, or may continue from one to several days, after which, little by little, respiration is re-established, the pulse returns, and the circulation brings new life into the limbs: after such a crisis the subject may awake immediately, or may continue to sleep.

A condition which is allied to that of hysterical lethargy or trance has long been known as *catalepsy*, a term to which, however, so many meanings have been attached as to give rise to an almost hopeless confusion in literature. In its correct use it signifies a form of morbid sleep which is characterized by loss of consciousness and of voluntary motion, and by a peculiar condition of the muscles, so that the body or the limbs take without resistance any position in which they are placed, and preserve without apparent effort such position for an indefinite time. In some cases of insanity and in other neuroses the muscles pass into a state somewhat resembling that of catalepsy. These are not, however, instances of catalepsy, to which, in truth, the loss of consciousness is as necessary as is the peculiar condition of the muscles. To designate muscular symptoms resembling those of catalepsy occurring in other conditions the term *cataleptoid* may well be used.

Catalepsy generally develops gradually, but it may come on abruptly as the result of a powerful emotion. As an instance of such sudden development may be cited the case recorded in 1415 by Lafaille (*Annales de Toulouse*, 1st part, 1415) of two Gray friars who, during a sermon on the Passion, were struck immovable in the attitude of devotion. Usually the cataleptic status is preceded by lethargy, convulsions, or other pronounced hysterical symptoms. It lasts from two hours to a day, and may

continually recur, so that the patient is said to be cataleptic for many days or even for weeks. The facial expression may be that of apathy; in some cases it is that of devotion, of rage, or of whatever passion the subject was in at the time of the fixation of the muscles. The eyes are wide open, with quiet lids. The body is motionless, in the posture in which it has been placed or in which it has settled during the arrest of active motion. There is no power of voluntary movement, but the limbs are not rigid or contracted. When taken hold of, they bend with the plasticity of wax. In any position in which the body or limbs are placed they remain for a long time, and Berger (quoted by Barth) is said to have seen the most bizarre and difficult attitudes steadily maintained for seven consecutive hours by a young cataleptic woman who was constantly under observation. During the whole of the cataleptic state there is complete anæsthesia of both the common and the special senses, so that the most violent irritations of the skin produce no reaction. Respiration is regular, the pulse maintains its normal rhythm and rate, and the general bodily functions appear to go on unaffected.

A condition resembling that of trance or catalepsy, in which consciousness is preserved, although the patient has no control over the voluntary movements, may develop as the result of a severe acute disease or from other cause. It is affirmed by Barth that in some cases which in other respects entirely resemble those of catalepsy the patient after coming out of the condition has recited all that occurred during the crisis. To those cases of lethargy in which consciousness has been preserved the term *lucid lethargy* has been applied. If in a lucid lethargy the death-like condition spoken of on page 394 has developed, the patient may be thought to be dead. There can be little doubt that under these circumstances premature burial of a conscious person has occurred. In my childhood I well knew an old and esteemed minister of the Society of Friends who, in one of the epidemics of yellow fever, after an attack of the disease, passed into this condition and was prepared for burial. Although perfectly cognizant of his danger, he could give no sign, until by a supreme effort he succeeded in making some slight movement of the eyelids, which indicated life.

If in insanity a cataleptoid condition appears, the true character

of the apparent catalepsy can usually be determined by noticing that the body and limbs when placed in bizarre positions show tremors or other evidences of fatigue. In catalepsy the limb when it falls falls like a wax arm which has been bent and slowly straightens by its own weight.

According to my thinking, it is not at present possible to arrange the various cases of morbid somnolence into symptomatic groups which can be distinguished from one another by the symptoms. In a previous chapter choreic movements were shown to be due to a peculiar condition of the ganglionic nerve-cells, capable of being produced by various causes. It seems to me that it must also be considered that the condition of morbid sleep is due to an altered nutrition of nerve-cells connected with conscious life, which altered condition cannot always be recognized by the microscope, and may be produced by various causes. The parallel between morbid somnolence and chorea is further evinced by the fact that the best classification that we can make of it is etiological. It will at once strike the reader that most of the groups which are separated in the following scheme are represented in chorea:

Group first, those cases in which the unconsciousness is due to a distant reflex irritation.

Group second, those cases in which it is an outcome of a peculiar nervous condition of unknown nature, to which the name of narcolepsy is applied.

Group third, those cases in which it is hysterical.

Group fourth, those cases in which it is connected with insanity.

Group fifth, those cases in which it is due to an organic disease of the brain.

Before taking up the consideration of these etiological groups, it is necessary to call attention to the fact that epileptic attacks may closely simulate morbid sleep.

Epileptic Sleep.—A sleep of some hours' duration out of which the patient cannot readily be awakened is a portion of a typical epileptic attack. In rare cases the whole epileptic paroxysm may be comprised in a sleep which may be prolonged for several days. The following case, reported by Surgeon M. Chabert (*Receuil de Mémoires de Médecine et de Chirurgie militaires*, 1867, vol. xviii.

p. 16), was probably epileptic. A soldier, twenty-six years of age, suddenly disappeared, and was marked as a deserter. Eighteen days later he was found deeply buried in a pile of straw lying in an out-house, the door of which is asserted to have been locked for seven days. The man was completely unconscious and apparently dead. The face was deadly pale, with dilated pupils, the limbs were relaxed, the surface was cold and without sensibility, the respirations were completely suspended, and even on auscultation no evidence of cardiac action could be made out. After half an hour's work with frictions, external heat, etc., an inspiration was taken: in a short time respiration and circulation were re-established, and on the next day the man was conscious. Inquiry showed that at the age of twelve years the patient had been seized with a furious migraine, had disappeared, and was found twenty-four hours afterwards asleep in a granary. Two months later he had a second similar attack. In the twelve or fourteen years after this he had seven or eight attacks, during which he would be deprived of consciousness for several days.

Another case, which may have been hysterical, but possibly was epileptic, is that recorded by Dr. Marduel. The soldier was admitted to the Military Hospital of Lyons on the 21st of March, 1870, profoundly unconscious and insensible, but with a full regular pulse of eighty-four, and calm respiration. In spite of the use of cold affusions and violent electrical currents, the sleep continued for seventy-four hours. Subsequently the patient had another attack of sleeping, during which there was a violent convulsion. The fact that in one of these sleeping periods there was general hyperæsthesia of such character that touching of the skin would immediately produce a violent tetanus or opisthotonos, points towards hysteria.

Reflex Unconsciousness.—It is well known that in poisoning by gastro-intestinal irritants stupor or insensibility may be so pronounced as to mask the ordinary symptoms. I have seen this in a case of poisoning with Paris green. I was once called to a child who was in profound insensibility, with very alarming collapse, apparently without cause, but who, I shortly learned, had eaten stale cream puffs a few hours before. By the use of a hot bath and emetics consciousness was soon restored. The following cases may be cited as instances of reflex unconsciousness.

In the *Boston Medical and Surgical Journal*, 1853, xlix. 363, is reported the case of a patient who, as the result of the ingestion of indigestible food, passed into a semi-comatose condition, in which he remained for two weeks and then died. At the autopsy the lesions found were severe inflammation of the small intestines and tubercular degeneration of the abdominal glands. A more satisfactory case is reported by Dr. Katerbau (*Magazin für Gesammte Heilkunde*, Berlin, 1825, p. 157); a seventeen-year-old Jewess, who had slept for four days and nights, under the influence of medicine passed a knot containing twenty-four round worms, and immediately awoke.

That the reflex sleep may take the form of catalepsy is shown by a case reported by Dr. Mayer (*Ann. de la Soc. Méd. d'Anvers*, May, 1863), in which a boy, nine years old, for five or six consecutive days had a diurnal attack of catalepsy lasting several hours, and was at once cured by the expulsion of a large number of intestinal worms.

Narcolepsy.—The cases of morbid sleep which are here grouped together under the name of narcolepsy vary in the intensity of their symptoms from drowsiness to a sleep which ends in death. It is most probable that the cause of the sleep varies, and that several distinct affections are represented in the group. The best, however, that can be done at present is to separate the cases into three sub-groups, which are not very clearly distinguishable and indeed are probably closely connected by intermediate cases. In the first of these groups the subject passes many hours in what seems to be ordinary slumber. In some cases the sleep comes on daily, in others at longer intervals. In some instances there is a perpetual drowsiness, in others the patient when awake is not sleepy. As illustrating these various facts I cite the following cases.*

Dr. J. W. Gloninger reports in the *American Medical Record*, vol. v., 1822, a case in which a man gradually passed into a condition in which he was excessively drowsy all the time, perpetually falling asleep when at work, and habitually spending eighteen hours out of every twenty-four in profound slumber. Dr.

* For a large collection of cases of morbid sleep, see paper by Dr. Dana, *Journal of Nervous and Mental Diseases*, April, 1884.

Hanfield Jones (*Lancet*, January, 1870) details the history of a porter who was never free from drowsiness, falling asleep at all times, and habitually spending fourteen hours of the twenty-four in sleep. Dr. T. Brady (*Medical Observations and Inquiries*, London, 1867) records the case of a woman who for many years had slept eighteen hours a day, except four months in one year, during which she was like other people, and twenty-one days in another year, when she had a tertian fever and slept not more than two hours at a time. This woman could not be aroused out of her sleep. G. Ballet reports (*Revue de Médecine*, ii., 1882) the case of a wine-merchant who, when three years old, had an attack of lethargy lasting seventeen days after typhoid fever, and at the age of twenty-six began to suffer from excessive drowsiness, which caused him to fall asleep in all sorts of places and at all times, although his nightly sleep was very long and profound. Dr. Rudolph (*New Orleans Medical and Surgical Journal*, 1883, xi.) tells of a young ranchero who, after having been troubled by excessive somnolence during the day, had spells of sleep lasting from twenty-four to forty-eight hours.

The second class of cases comprises those in which the paroxysms of sleep come on at irregular intervals and continue for days. As an instance of this form of narcolepsy I cite a case reported by Dr. Outrepont (*Neue Zeitschrift für Geburtskunde*, 1844). A Jewess shortly after her marriage fell into a prolonged sleep, which had ever afterwards recurred periodically. The average length of the sleeping period was five and a half days, the longest time that she had ever slept being seven days. The intervals of wakefulness lasted from two to twenty days, during which time she did not sleep at all, or had only a very little restless slumber. The sleep would come on suddenly, sometimes in the night and sometimes in the middle of the day. At the end of the first twenty-four hours she would awake with a very dry mouth and put out her tongue as though she wanted a drink, when an attendant would give her fluid nourishment, after which she would immediately go to sleep again. She appeared to have no consciousness of this brief awaking. She could not be aroused, but would awake spontaneously and suddenly in a very weak condition. The pulse during the sleep was about sixty-six, regular; the respirations were so feeble that the movements could scarcely

be observed, and the temperature was normal.. Neither the urine nor the fæces were passed during sleep. The pupils were normal, but did not respond to light. Dr. W. G. Gimson (*British Medical Journal,* 1863, i. 616) reports a case in which a man after a severe cold had attacks of profound sleep. The sleep would last from twelve to twenty-four hours; the respirations were eighteen, quiet, the pulse sixty-four, regular, feeble; skin warm, hands and feet cold; he could not be awakened, and waked suddenly at last. At the time he was seen by the doctor the man passed forty out of forty-eight hours in sleep; once he slept eighty-four hours. He never took food nor had a passage from the bladder or the rectum during sleep. When awake the mental action was as good as ever.

A third class of cases is that in which the sleep comes on without apparent cause, and becomes more and more profound until the patient dies. Thus, in a case reported by Dr. S. Weir Mitchell to the College of Physicians, a woman suddenly became giddy and fell insensible; from this condition she soon aroused, but three days later she passed into a condition of sleep, during which she could be momentarily aroused and would answer simple questions. For eleven days she remained in this state, with some convulsive movements, and then died quietly. Careful post-mortem examination, with a microscopic study of the brain, failed to detect any cause of death. Some of the cases of the present group are probably instances of intense cerebral congestion, and might be relieved by venesection. Thus, Dr. Charles S. Spilman reports in the *Transylvania Medical Journal* the history of a boy, fifteen years of age, who, after the death of his father, fell into a condition of profound sleep, with occasional slight convulsive movements and a slow, laboring pulse. He could not be aroused; but after seventy-two hours of sleep forty-four ounces of blood were taken, and the boy at once awakened. In some fatal cases of apparent narcolepsy distinct lesions after death have been found. Thus, Dr. Haine reports (*Gazette des Hôpitaux,* vol. xlii., 1869) the case of a girl, nineteen years of age, who died after a profound sleep which had come on suddenly and continued for fifty-six days. At the autopsy a small and very circumscribed spot of softening was found in the cerebrum.

Hysterical Sleep.

The most common variety of morbid sleep is that which is connected with hysteria. The symptoms usually take the form of lethargy or trance, with or without catalepsy. True narcolepsy may, however, be closely simulated, the patient being continually drowsy, and falling asleep at all times, but passing only the nights in profound slumber. The presence of other hysterical symptoms commonly betrays the nature of the somnolence. As illustrating the manner in which the various symptoms of hysteria are usually intermingled, I cite the following case reported by W. T. Gairdner (*Brit. Med. Journ.*, October 30, 1875), in which a girl, after hysterical fits and hysterical chorea, suddenly became unconscious, with wide-open eyes and dilated pupils, at the same time speaking and singing incoherently, apparently as the result of hallucinations and delusions. This continued for about two weeks. A month or two later she fell into a deep sleep, which lasted eight days with an interruption of a few moments. During this sleep she could not be aroused, and had no passage from the bowels or the bladder. Some months later she had a second eight-day sleep: after awaking from this she passed into a condition in which she slept persistently, unless aroused, when she would get up and dress herself, but would remain awake only so long as she was in active exertion.

A case of hysterical lethargy which shows the close relation between it and insanity is recorded by Dr. F. R. Mueller (*Journal der Praktische Heilkunde*, 1829, vol. lxviii.). A young woman who had had slight melancholia was suddenly seized, whilst at church, with intense sleepiness, sat down on the door-step, went to sleep, was after a time carried home, and slept, with very brief interruptions, for four years, three months, and sixteen days. Once during this period she was awake eighteen days, sleeping naturally at night, and seeming like herself. Her uninterrupted sleeps lasted from forty-eight hours to a week; her waking periods were at first only ten or fifteen minutes, but afterwards several hours. During the sleep she would lie quietly upon her back, never altering her position, with her hands folded over her abdomen. The skin was parchment-like and dry; the eyelids were closed, with the eyeballs, when exposed, divergent; the face was deadly

pale, exeept the lips, which were red; the pulse was slow and regular, the breathing slow and verv light; the insensibility of the skin was eomplete. She was mueh emaciated, and the temperature seems to have been subnormal. There was habitual stiffness of the museles, which, however, could be readily overcome, and in the earlier months of sleep she had at times distinct cataleptie symptoms. Often her somnolenee went off gradually.

The relation of hysterical sleep to the eondition known as hypnotism is so close that it seems to me proper at this place to eonsider briefly the subjeet of hypnotism. To discuss all the phenomena of the state would require mueh more spaee than is permissible.*

Hypnotism.—By eausing a suseeptible person to fix his eyes steadily upon a bright objeet, as a button, or by pressing the eyelids upon the eyes, or by other suitable procedure, the condition of hypnotism is produced. In its most typical form it is eomposed of three stages: first, catalepsy; second, lethargy; third, artificial somnambulism.

In catalepsy the subjeet appears as though petrified in his position. The eyes are fixed widely open, with dilated pupils and an insensible cornea. With rare exeeptions, the general surfaee of the body is insensible to pain, but the special senses retain their activity. The extremities are supple, but when bent maintain for a great length of time any position into which they may have been plaeed. In this state (and still more markedly in the condition of lethargy) *paradoxical contractures* may be produced. They are developed by so flexing or bending a part as to throw the musele into sudden and complete relaxation, when it immediately passes into a eondition of severe tonic spasm. The contraetures may also be developed by striking the tendons, or even by rubbing the belly of the musele. A peculiar phenomenon which occurs during the eatalepsy is that if the body or limbs are put into a position expressing some emotion, the faee takes upon it an expression corresponding to this emotion, and the whole individual seems overwhelmed by emotional excitement. Thus, if the arms are thrust forward and the hands raised as though

*For details the reader is referred to the work of Ferdinand Bottey, *Magnétisme animale*, Paris, 1886, as an excellent treatise upon the subject.

pushing away or shoving something out of sight, horror and fear gather upon the countenance, and the whole body seems to be recoiling from some dread object; if the person be placed in the attitude of prayer, the expression will become one of intense devotion; if the posture be that of combat, rage will be developed.

During the lethargic state there is complete relaxation of the whole muscular system: the head falls upon the shoulders, and the limbs are absolutely flaccid, and when raised drop as though dead. The eyes are closed, and frequently both the lids and the balls tremble constantly. The skin is insensible, so that pinching, sticking with needles, or other irritation provokes no response.

The somnambulistic condition occurs in two forms,—one with the eyes closed and the other with the eyes open. The appearance of the person who is in somnambulism with closed eyes is precisely what it has been during the state of lethargy, but now on the word of command the somnambulist rises, marches, and does whatever he is commanded. The insensibility of the skin remains, but the special senses are awake, and even much more acute than normal, so that the subject will be able to read in a darkened room, to hear sounds inaudible to others, or to recognize odors not perceptible by others. There is also in some cases exaltation of the intellectual faculty. Thus, a young man, a student of mathematics, during an hypnotic state, solved with elegance and rapidity problems in trigonometry which during his natural condition he had essayed in vain. There is also a revival of the memory of facts apparently long since forgotten, and even an expression of remembrances hitherto unknown to the consciousness of the individual when awake.

The phenomena of somnambulism with open eyes differ from those of somnambulism with closed eyes in that instead of the subject being absolutely automatic he is full of an unconscious activity, and when left to himself moves restlessly hither and thither; at times he offers considerable resistance to the will and commands of the experimenter. The eyes are wide open, with the sight fixed, as in catalepsy, upon vacuity, or there may be a wild expression. Spontaneous hallucinations appear frequently to rise within the brain of the somnambulist and to find expression in both word and deed.

Sleep in Insanity.

Although true stupor may follow upon or be connected with pronounced evidences of mental aberration, yet in a large proportion of cases it is apparent rather than real. If an insane patient lie in bed absolutely still and inert, with closed eyes, giving no response to the loudest questioning and making only a feeble and slow resistance to personal violence,—or if, with head bent forward, joints flexed, and face frozen into an immobile apathy, he sit motionless in his chair,—he seems to be lost in unconsciousness, but none the less may he have knowledge of his surroundings and of his sorrows. This lethargy may be the direct result of an intense emotion or of delusion, and not be consciously assumed, but not rarely it is put on for a definite end, and maintained with a tenacity of purpose which defies detection even during the intoxication caused by ether or by alcohol. In a majority of cases, however, an assumed stupor can be detected by the use of intoxicants. In many cases it is impossible to penetrate the veil and to determine why the insane person keeps up for months an absolute silence and passivity; but the occasional revelations made by patients after they recover their reason show that a delusion may act very directly. A man believes that he has received commands from the Almighty to do as he does, and battles for his eternal salvation; or he conceives that his attendants are conspiring against him, and will do him great evil if once they are assured he is alive. In some cases the lethargy is the result of an overwhelming emotion produced by the delusion. The man about to be devoured by foul beasts or by the flames of hell is dumb through fear, or, as the German alienists say, is thunderstruck. Occasionally the insane sleeper is convinced that he is dead, and by this delusion his will is so far paralyzed that it is unable to act, and the man really cannot move, although the lower nervo-muscular apparatus is intact.

Toxæmic Sleep.—The only forms of toxæmic sleep which require discussion are those which arise in chronic Bright's disease and in diabetes mellitus. The symptoms of uræmia have already been sufficiently discussed, and it only remains to consider *diabetic coma.*

Coma occurs during the course of diabetes in several forms.

It may come on late in the disease as the result of secondary organic alterations in the brain itself. In another class suddenly the strength gives out, the pulse becomes very rapid and weak, the extremities grow cold, and in a very few minutes or hours the patient sinks into a syncopal stupor, which ends in death. The cause of these symptoms is sudden failure of a heart whose muscle has degenerated. Neither of these two classes of cases are entitled to be called diabetic coma. True diabetic coma may occur at any time or stage of the disorder. It is usually preceded by a train of nervous symptoms, which, with the coma, are now believed to be due to the presence in the blood of a substance produced by the decomposition of the sugar. This substance is supposed by some authorities to be aceto-acetic acid, and seems to be at least an acetone-producing principle. For this reason the name of *acetonæmia* has been given to diabetic coma. The peculiar odor of acetone can in most cases be detected in the breath, the urine, and the perspiration, whilst usually there is sufficient acetone in the urine to strike a Burgundy-red color with a solution of chloride of iron.

According to Prof. Frerichs (*Ueber den Diabetes*, Berlin, 1884), there are two distinct forms of diabetic coma. In the one variety, after great weakness, gastric disturbance, vomiting, diarrhœa, and perhaps some local inflammation, as a carbuncle or a bronchitis, there develop headache, restlessness, delirium, excessive anxiety, dyspnœa, with very deep expirations and inspirations, and with or without evident cyanosis, fall of temperature, great rapidity and feebleness of the pulse, somnolence, and finally coma, which ends in the majority of cases in death after from one to three days. In the other form of diabetic coma, whilst the patient is apparently in good bodily condition and has no dyspnœa, headache, staggering gait, and somnolence suddenly come on and end in a coma, which invariably proves fatal in a short time.

ORGANIC STUPOR AND COMA.

Organic diseases of the brain of which stupor or coma is a prominent symptom can best be studied for the purpose of diagnosis by dividing them into those which are accompanied by marked headache and those in which headache is wanting. These groups I shall respectively note as Group First and Group Second.

Group First. Organic brain-diseases in which headache and stupor are prominent symptoms are naturally divided into two sets, specific and non-specific.

Non-specific Stuporous Affections.

Brain-Tumor.—Stupor is liable to develop at any time during the course of a brain-tumor, although it is in no sense a characteristic symptom of such affection. In the last stages of brain-tumor, when the surrounding cerebral substance is undergoing softening, or when by pressure or progressive disease the important vessels are interfered with, stupor or profound coma is very common. The recognition of such a coma must depend upon the previous study of the case. Of different import is the stupor which occasionally develops from time to time in the earlier stages of the brain-tumor, and is not dependent upon severe structural lesions of other portions of the brain than those immediately implicated by the growth, but to a general cerebral congestion. Such stupor may or may not be accompanied by convulsions. It frequently comes on rapidly, and may in the course of a few hours pass off, or may remain several days and then subside. I have seen a patient with a gliomatous tumor who had been for several days absolutely comatose, passing the discharges involuntarily, and thought to be dying, a few hours later walk to the clinic-room in a distant portion of the hospital.

Meningitis.—A second cause of organic stupor and coma is inflammation of the meninges. Acute meningitis habitually ends in coma, and any time during the course of a chronic meningitis the symptom may be developed. The significance of coma occurring during an acute or even a chronic meningitis can scarcely be mistaken. The detailed discussion of the symptoms of acute and chronic meningitis will be entered upon in the next chapter.

Pachymeningitis Hæmorrhagica.—Pachymeningitis hæmorrhagica is a disease in which there is chronic inflammation of the dura mater, with the formation of a bloody growth or tumor, due to or connected with repeated hemorrhages into the part. It is essentially an affection of old age, or of persons who, from syphilis, scorbutus, or other constitutional dyscrasia, have degeneration of the vessels. It also occurs as a secondary affection in dementia paralytica, brain-atrophy, hydrocephalus, etc. The head-

ache is usually severe and throbbing, in most cases is not accurately localized, and often occurs in furious paroxysms, especially at the time when fresh hemorrhages take place. The motor disturbance may show itself in paresis, or in localized muscular movements, or in general epileptic convulsions. A shifting hemiplegia, which is now on the one side and now on the other, is not uncommon. Muscular twitching with subsequent rigidity may accompany or follow the shifting palsy, and permanent hemiplegia with contractions may develop. Conjugated deviation of the eyeballs is not infrequent, but, as the hæmatoma is almost invariably on the vault of the cranium, the ocular and other basal nerves are rarely, if ever, involved. If facial palsy happen, it will take the form that is characteristic of central brain-disorder. Psychical disturbance is very common, and vertigo is frequent. The pupils may be contracted and insensible to light, but when the cerebral compression is marked they dilate. During conditions of cerebral compression the pulse may be slow, but the pulse-rate varies almost indefinitely throughout the disorder. Drowsiness with an habitual excess of sleep is rarely wanting in cases which do not run a very rapid course. The stupor may be prolonged, but more frequently it comes and goes as the cerebral congestion varies. Profound coma is usually developed when pressure occurs from renewed hemorrhages, and in the later stages of the disease when the cerebral substance is undergoing alterations in the neighborhood of the lesion.

In a large proportion of cases pachymeningitis is not recognized during life, and the diagnosis may be impossible. If the patient die in an early hemorrhage the symptoms will be simply those of apoplexy preceded by a more or less pronounced headache. In prolonged cases with characteristic symptoms the nature of the affection should be made out. The symptoms may resemble very closely those of tubercular meningitis, which is, however, an affection of children : if the patient be past middle life and be free from tubercular disease in other portions of the body, the diagnosis of pachymeningitis hæmorrhagica would be justified,—a diagnosis which would be greatly strengthened by finding degeneration of the vessels in other portions of the body. Choked disk, in one or both eyes, is frequent, and the coma in pachymeningitis occasionally has remissions and exacerbations closely resembling those which sometimes occur in brain-tumor. The

affection is therefore liable to be confounded with brain-tumor; but the age of the patient and the peculiar drowsiness which occurs between the comatose conditions usually render the diagnosis possible. Fürstner has called attention to the temperature of the body as a means of diagnosing between pachymeningitis hæmorrhagica and a cerebral apoplexy. He believes that in the meningeal hemorrhage rise of temperature is not preceded by a fall, whilst in intra-cerebral hemorrhage such fall usually, although not invariably, occurs. (*Archiv für Psychiatrie und Nervenkrankheiten*, 1878, Bd. viii. pp. 1–31.)

Specific Stuporous Affections.

Syphilitic Coma.—The ordinary lesions produced by cerebral syphilis are meningitis, localized or diffused, and degeneration of the cerebral vessels. Either of these changes may give rise to somnolence or to profound coma. Such coma does not, however, in its symptoms conform to a regular type, as described by Dr. Julius Althaus (*Medical News*, vol. xlix. p. 428), but varies greatly in its manifestations. In the wards of the Philadelphia and University Hospitals the affection is so frequent that, although at least sixty per cent. of the cases recover, I have seen three die in one week; and studies made chiefly in those hospitals lead me to divide syphilitic coma for the purpose of discussion into several varieties.

The first and least common form may be known as *coma foudroyant*, or *fulminating coma*. The symptoms in such cases may appear to develop suddenly in the midst of good health, but I believe that close examination will show that headache, vertigo, or some other indication of organic brain-lesion has always preceded the violent attack. I do not believe that acute syphilitic meningitis or an acute coma develops as a primary lesion or as a primary symptom: both the lesion and the symptom are preceded by the formation of the gummatous tumor, or by pronounced degeneration of the vessels. It is certain, however, that the structural disease may be essentially latent, and the attack appear to come on abruptly in the midst of health. An acute fulminating syphilitic coma might theoretically depend upon the obliteration of the cerebral vessels by embolus or thrombus. In such case the symptoms would be those of embolism or thrombosis from other than

specific causes. The alterations in the cerebral vessels produced by syphilis are slowly progressive, and, although they not rarely end in cerebral softening with its accompanying stupor and coma, the symptoms in all the cases which I have seen have developed slowly; the blood-current seems to be gradually shut off. Foudroyant or fulminating syphilitic coma is an outcome of a gummatous inflammation or growth.

The stupor may or may not be accompanied by delirium or by convulsions. A man about thirty years of age, whom I saw in consultation, thought himself in perfect health, but became very drowsy about the middle of an afternoon, and, going to the back of his store, fell asleep. Being found in this condition, he was aroused, and with assistance got up-stairs to bed. Very shortly afterwards he became comatose, with delirious outcries and furious convulsions. In a case reported by Dr. J. A. Ormerod, a man who had been in good health, with the exception of headache, awoke one morning in a semi-delirious condition, and for three days slept steadily, arousing only for meals: after this there was impairment of memory and of the other mental faculties, but there were no more marked symptoms.

In the cases reported by Dr. Althaus the coma developed rapidly and quietly, and in several instances during sleep, so that, although local or general convulsive symptoms may be pronounced in fulminating syphilitic coma, the patient may be completely quiet and relaxed. Under these circumstances the symptoms are simply those of profound coma. There is nothing in the coma itself which will enable us to distinguished its specific source. Hemiplegia or evidences of local palsy are usually wanting, but I have no doubt that it is possible for them to be present: in the case which I have just mentioned, after recovery of consciousness partial hemiplegia was very noticeable. The condition of the pulse varies, as it does in coma from other causes. The rate may fall far below normal, or the pulse may become rapid and fitful, or it may be hard and wiry: it may be large with high tension, or it may be large and soft. The cause of the coma is, I believe, congestion of the brain, entirely parallel to that which occurs in cases of non-specific cerebral growths. The recognition of the fact that the symptoms are not peculiar, and are due to a secondary congestion of the brain, is very important,

because it leads to the practical conclusion that the first treatment of such a case must be precisely that which would be used for the relief of similar symptoms due to non-specific brain-lesions. In some cases life has been saved only by free venesection. After the acute symptoms have been subdued, very active specific treatment should be instituted.

The second variety of syphilitic coma develops gradually. The patient sits all day long or lies in bed in a state of semi-stupor, indifferent to everything, but capable of being aroused, answering questions slowly, imperfectly, and without complaint, but in an instant dropping off again into his quietude. In other cases the sufferer may still be able to work, but often falls asleep while at his tasks, and especially towards evening has an irresistible desire to slumber, which leads him to pass, it may be, half of his time in sleep. This state of partial sleep may precede that of the more continuous stupor, or may pass off when an attack of hemiplegia seems to divert the symptoms. The mental phenomena in the more severe cases of somnolency are peculiar. The patient can be aroused,—indeed, in many instances he exists in a state of torpor rather than of sleep; when stirred up he thinks with extreme slowness, and may appear to have a form of aphasia; yet at intervals he may be endowed with a peculiar automatic activity, especially at night. Getting out of bed; wandering aimlessly and seemingly without knowledge of where he is, and unable to find his own bed; passing his excretions in a corner of the room or in other similar place, not because he is unable to control his bladder and bowels, but because he believes that he is in a proper place for such acts,—he seems a restless nocturnal automaton rather than a man. Apathy and indifference are the characteristics of the somnolent state; yet the patient will sometimes show excessive irritability when aroused, and will at other periods complain bitterly of pain in his head, or will groan as though suffering severely in the midst of his stupor,—at a time, too, when he is not able to recognize the seat of the pain. I have seen a man with a vacant, apathetic face, almost complete aphasia, persistent heaviness and stupor, arouse himself when the stir in the ward told him that the attending physician was present, and come forward in a dazed, highly pathetic manner, by signs and broken utterances begging for something to relieve his head. Heubner

speaks of cases in which the irritability was such that the patient fought vigorously when aroused: this I have not seen.

After some days of excessive somnolence and progressive deepening of the stupor, or sometimes more rapidly, the victim of cerebral syphilis may pass into a condition of profound coma, out of which he cannot be aroused, and during which his fæces and urine are either not passed at all or are voided involuntarily. This condition of coma may end in death; but even when the symptom seems most serious the patient may gradually recover, slowly emerging from coma into stupor, and from stupor into wakefulness and normal life. I have several times seen excessive somnolence, lasting four or five months, during most of which time the patient was actually comatose, more or less thoroughly recovered from. In most of these cases hemianopsia, or motor palsy, or altered mental power, has remained to show that the brain had been permanently damaged. On the other hand, even in extreme cases the recovery may be complete.

Syphilitic stupor ending in death usually puts on symptoms exactly resembling those of advanced brain-softening, to which, indeed, it is in most cases due. I have made three autopsies on such cases: in one there was symmetrical purulent breaking down of the anterior cerebral lobes; in the second, softening of the right frontal and temporal lobes, due to pressure of a gummatous tumor; in the third, softening and breaking down of the brain in the region supplied by the middle cerebral artery, probably as the result of an arrest of circulation.

Group Second. The organic brain-diseases which produce stupor but are not associated with headache are not very numerous: prominent among them is dementia paralytica, in which disease, however, the stupor does not come on until very late in the affection. In all forms of cerebral softening stupor or coma is finally developed, and if such softening be due to disease of the blood-vessels, to cerebral sclerosis, or to other affection which does not involve the brain-membranes or markedly increase the blood-pressure in the brain, headache is usually wanting. A rare but important affection, of which stupor is the most prominent symptom, is one which has not been heretofore generally recognized as a distinct disease, although cases of it have been reported.

There is so much unity of structure in the ganglionic cells throughout the nervous system that it can hardly be otherwise than that a pathological process which affects one set of these cells shall find its parallel in the disease of other cells. A sclerosis may attack any portion of the white matter of the nervous centres, and in like manner I believe that the peculiar degeneration which occurs in poliomyelitis may assault the nervous cells of the cerebrum. Under the circumstances, loss of function, with or without evidences of primary irritation, must result. For this affection the name of *Polioencephalitis* is very suitable.

In some cases of poliomyelitis fever and other evidences of constitutional disturbances are pronounced in the beginning of the disease; but it does not follow from this that the changes in the cells are due to acute inflammation, much less have we any proof that the slow alterations which occur in the subacute and chronic forms of the so-called poliomyelitis are inflammatory.

It is probable that all these forms of poliomyelitis are represented in affections of the cerebral cortex; but the only cases of which I have knowledge in which the nature of the disease has been proved by post-mortem examination represent the subacute forms of the affection.*

The palsy and the trophic changes of poliomyelitis are evidences of loss of functional power. Symptoms due to the irrita-

* It is possible that the cases described by Dr. Adolf Strümpell (*Allgemeine Wiener Med. Zeitung*, 1884, 29) under the name of encephalitis of childhood may represent the acute form of poliomyelitis; but as in no case was an autopsy made, and as the symptoms closely resemble those of ordinary infantile spastic paralysis, the matter is open to much doubt, especially since Dr. Strümpell states that he has never seen the affection in its earlier stages, and has had to rely upon the statements of parents for the descriptions. According to these statements, the sickness commences with fever, vomiting, and convulsions, or, in some mild cases, with a short convulsion and slight febrile reaction. This stage is said to last from two days to a month, during which time the parents have noticed paralysis of one-half of the body or sometimes of a single extremity. The hemiplegia is never complete, the paralysis often affecting groups of muscles and constituting a multiple palsy. There are no trophic changes, and in many cases facial or ocular palsy shows that the nervous system is affected very high up. After the acute stage had passed, at the period when the cases were seen by Dr. Strümpell, there were marked contractures, with increase of the reflexes. In some cases there was athetosis. Epileptic attacks were not infrequent, and not rarely there was marked mental degeneration.

tion of the ganglionic cells seem to have no place in the affection, unless, indeed, the convulsions which usher in the attack be looked upon as of such character, but these are probably cerebral, and sympathetic local spasms do not occur, as should be expected if there were any persistent irritation of the motor ganglionic cells. In like manner the symptoms of polioencephalitis, at least of the subacute cases, are the outcome of failure of function. They may be summed up as failure and perversion of intellection, persistent stupor, muscular relaxation, anæsthesia, and, in cases which do not recover, death in a stuporous dementia. It is well known that in poliomyelitis partial or even complete recovery sometimes takes place, although the symptoms have seemed desperate, and it is probable that some of the cases of persistent stupor occurring among the insane, followed by more or less imperfect recovery, are instances of polioencephalitis.

The following case is reported by Dr. Legrande du Saulle (*Gazette des Hôpitaux*, 1869, xlii. 505). I conceive it to be a characteristic instance of subacute polioencephalitis. A man, aged thirty-two years, became melancholic during June, 1868. On the 10th of September he went into a profound sleep. The extremities were rigid, the respiration rapid, the pulse seventy-two, and general sensibility very obtuse. About a week after this, general muscular relaxation developed, the cutaneous anæsthesia became complete, and intestinal inertia very pronounced. The respirations were from twenty-four to thirty-two a minute. By October the temperature of the body had fallen to ninety-six degrees, emaciation had become extreme, and the bowels were opened only once in ten or twelve days. On the 4th of November, after a blister to the head, he suddenly cried out, "My God, my God, have pity on me, for I am about to die!" but a moment afterwards relapsed into stupor. The pulse during October was forty-four a minute, but with his general condition began to improve, and by the close of November the pulse was nearly natural in frequency and the intestinal action about normal, but the stupor persisted. The urine was normal, except that it contained some excess of uric acid. Intoxication with alcohol, ether, and hasheesh failed to elicit any sign of mental life, as did also the efforts of a *magnétiseur*. The optic papilla was very pale, and opaque from serous transudation. The man died of pneumonia in the latter part of

March. A thorough autopsy showed that the cerebral gray matter had changed color to a pale gray, through which were scattered sharply-defined islets of excessive vascularity. The nerve-cells of the cerebral convolutions had almost completely disappeared.

ACCIDENTS OF SLEEP.

Under the head of accidents of sleep I propose to consider, briefly, first, certain curious symptoms which have no connection with dreaming; secondly, certain states which are closely connected with dreaming.

In the first division of the present subject the disturbances are chiefly sensory. The most important of them is that to which the name of *sense-shock* has been given by Dr. Mitchell. It is most frequent in hysterical women, but does occur in men, especially in those of a neurotic temperament who are overworked. It is usually felt at the time when the subject is passing from waking to sleep. A sensation like an aura rises from the feet, or, more rarely, from the hands, and passes upward to the head, where it disappears in the sense of a blow or shock, or of a bursting in the head: not rarely at the time of the explosion the patient hears a loud noise, or sees a vivid flash of light, or perceives a strong odor. In some cases two or even more of these sensory manifestations are present together. There is no loss of consciousness, and any motor symptoms which may occur are the outcome of the overpowering terror which is felt during the crisis and is sometimes manifested by a shriek. Occasionally a number of these shocks follow one another at short intervals. The paroxysms may occur during the daytime. These attacks may be excessively annoying, but they have no serious significance, and are to be looked on as hysterical.

A sleep-symptom which Dr. Mitchell states that he has seen in Duchenne's disease is that to which the name of *night palsy* or *nocturnal hemiplegia* has been given,—a name which seems to me improper and misleading, as the symptom is not connected with loss of motor power, and, in my experience at least, is never of serious import. I have frequently seen it in hysterical or neurotic women, especially at the time of the menopause, but never in organic nervous disease. It consists simply of a feeling of numbness in one or more extremities of the body when the sleeper awakes.

The most common seat is one arm; but the symptom may be hemiplegic, or may affect the whole body. It certainly is not the result of lying upon the part, nor is it any indication of heart-disease or of organic nervous disease. It appears to me to be simply one of the numerous hysterical symptoms whose exact nature cannot be explained.

Somnambulism.—Somnambulism is defined by Dr. H. Barth (*Du Sommeil non-naturel*, Paris, 1880) to be a dream with exaltation of the memory and of the automatic activity of the nerve-centres, combined with absence of consciousness and spontaneous will. It is common for a sleeper, be he either human or brute, to give evidence, by speech or by movement, of the dreams that are coursing through his brain. Such evidences may in the dog be no more than a bark, or an impotent running motion of the feet, or a wagging of the tail; whilst in the man restless tossing, movements of the hands, or muttered words may be the sole indication of what is going on within. A step beyond this, and the dreamer acts in accord with the drama which is being enacted in his imagination. Thus, a man strikes his wife in the belief that he is wrestling with burglars. Sometimes after such agitated movements, or in the midst of an apparently profound quiet repose, the sleeper rises from his bed, and, unclothed, or after first dressing himself, passes about his room, opens his door, goes out, or does other acts with continuous rapidity of movement. Every grade between the slightest dream-movement and the most active sleep-walking exists; but whenever a dreamer rises from his couch he may be said to be a somnambulist.

If the somnambulist be approached, his eyes will be found to be closed, or, if open, with the rest of the face they are impassible and without expression, paying no attention to the brightest lights, and appearing to have no power of sight in them: yet obstacles are avoided, narrow places passed through, feats of balancing performed, and numerous complicated movements made so perfectly that the by-stander can hardly persuade himself that the sleeper is not awake. When seized hold of, the somnambulist usually resists with vigor. Left to himself, after wandering for a greater or less length of time he returns to his bed, covers himself up, and sinks into the quiet forgetfulness of normal sleep.

In the milder forms of somnambulism it is sometimes possible

to turn the thoughts of the sleeper by speaking to him, and in obedience to a firm command he will return to his bed without awakening. Shaken a little strongly, or aroused with a dash of cold water, he awakens slowly, and in a little time is conscious of his environment.

In the more severe forms of somnambulism the paroxysm lasts for a considerable time, and during its continuance acts are performed which seem impossible to an unconscious man. Thus, the somnambulist will actively rehearse that which during waking hours occupies his thoughts and his acts. A parson will prepare his sermons, a student labor over his tasks, an artisan toil with his hands. Bourgarel (*Union Méd.*, 1861) records the case of a sailor who would rise from his hammock, wander about the vessel, climb the masts, and high above the sea go through the duties of the foretopman. Barth recounts the case of a student whom his comrade saw get up and go into his study and compose a piece of Latin verse, but who on the morrow was ignorant of all that he had done, and reported to his professor that through lack of time he had been unable to perform his allotted task. Sometimes the somnambulist will reproduce by word, gesture, or act scenes which emotional excitement has impressed upon the nervous organism. Such was the case of a young girl, cited by Barth, who would recount with detail in word and act a criminal assault from which she had suffered.

Even in the mildest forms of the affection a somnambulist may be led by his dreams to acts of violence, and in the severer paroxysms serious injury may result.

Barth quotes a case originally recorded by Alfred Maury, in which a husband attempted to throw his wife out of the window whilst dreaming that his house was on fire. M. Fodéré, in his treatise on medical jurisprudence, details a case related to him by a prior, who, going very late to bed one night, saw one of the brethren walk in his sleep up the entry, open the door of his (the prior's) room, and pass in. For a moment the somnambulist stood, with open, fixed eyes, and an expression of determined rage upon his face, and then marched to the bed with a drawn knife in one hand. Passing his unarmed hand over the bed, he seemed to feel the presence of some one in it, and then struck fiercely with the knife three times, forcing the blade through the bedclothes deep

into the mattress. After this, with an air and expression of great satisfaction on his face, he turned and went back to his own bed. The light of two lamps shone in the room, and apparently fell directly upon the eyes of the somnambulist, but elicited no response. The next morning the prior sent for the brother, who, on being urgently questioned, said that he had had a frightful dream the night before. He had dreamt that the prior had killed his mother, that her bloody ghost had appeared to him demanding vengeance, and that under its direction he, in a transport of fury, had forced his way into the apartment of his superior and killed him with a poniard. He ended his account by expressing the immense relief which he had experienced when he awoke and found that all was but a dream.

A case which ended more tragically was tried before the English courts, and was reported by Dr. Yellowlees in the *Journal of Mental Science* for October, 1878. The history was that the family of the prisoner, while he was still a mere lad, lived alongside of a rushing torrent, and that often he would arise in his sleep and go to the landing-place, and even into the water, loudly calling his favorite sister by name, feeling out with his arms as if rescuing her from drowning. Sometimes the water awoke him, and sometimes it did not, but after his efforts he would go quietly to bed. As his life went on, he became more and more liable to seizures of night-walking, which finally settled down into a common type. During his sleep terror would seize upon him, and he would start out of bed to escape or put aside the impending evil. In his dreams the house would be on fire, the walls would be crushing him, or his child would be falling down a pit, or still more frequently a wild beast had come into the room and was about to devour him: roaring frantically, and in an agony of fear, he would tear his wife and child from the bed and fiercely chase the wild beast through the room, throwing the furniture about, and striking wildly with any weapon that he could reach. On several occasions he had seized a companion by the throat and strangled him almost to death, under the idea that he was struggling with the wild beast. In some of these paroxysms he would hear and answer distinctly. One night he saw a large white beast fly up through the floor and pass towards the bed where the child lay: to save it he gripped it by the breast, and, roaring with terror, hurled

it against the wall with such force that it fell dead. That the paroxysm resembled an attack of epileptic delirium is apparent; and Dr. Echeverria (*Journal of Mental Diseases*, January, 1879) attempts to prove that the patient really suffered from nocturnal epilepsy. The fact, however, that the man could be awakened during the paroxysms indicates very strongly that the attack was not a pure epilepsy, which is confirmed in a measure by the total absence of epileptic phenomena during the daytime. The urinary incontinence which Dr. Echeverria speaks of as having been present and as evidence of the epileptic character was, according to the report of Dr. Yellowlees, essentially different from the incontinence of nocturnal epilepsy. It occurred only during childhood, and entirely independently of the paroxysm, and was simply the incontinence of a feeble, neurotic child.*

The so-called *night-terrors* of childhood, although frequently spoken of as a distinct affection, are, in truth, only a form of somnambulism, or, in rare cases, epileptoid seizures. Nothing is more common than for a young child to go in the night to its parents' bed, trembling with terror or weeping bitterly, with the statement that it has had a bad dream. Such a dream may be so vivid as completely to enchain the attention, and if at the same time there be outward manifestations of the overpowering emotions from which the child is suffering, a paroxysm of night-terror results. With screams and imploring calls upon its mother for assistance the child struggles and cannot be aroused or comforted, but at last slowly awakes, or, much more infrequently, falls again into peaceful slumbers. Very frequently, even during the paroxysm, the child shows terror of some one object: a cat, a dog, a white elephant, a monster of some kind, is indicated by its incoherent cries. In a large majority of cases night-terrors are not connected with any organic disease of the brain or with epilepsy,

* Medico-legally this case is of great interest. Put on trial for his life, the man pleaded, "I am guilty in my sleep, but not guilty in my senses," and the jury found that the man was unconscious of the nature of the act which he committed by reason of a condition arising from somnambulism, and that he was not responsible. The man certainly was not insane in the ordinary sense of the word, and just as certainly he was not responsible. These cases are so exceptional that I believe no country has as yet a law applicable to them. Probably the best that a court could do would be to consider the alleged criminal as having been temporarily insane.

and are of no more serious import than an attack of somnambulism. They usually depend upon some peripheral irritation: especially are they commonly the result of a gastro-intestinal irritation from undigested food. Hence they frequently follow heavy suppers, or overeating of some kind in the latter part of the day. Not rarely they occur during active dentition, and are relieved by cutting of the gums. In a few recorded cases the cause of the attacks has been intestinal worms. The overpowering emotion of the night-terror is sometimes the result of a fright during the day, as in the case reported by Meigs and Pepper, in which a child, who had been bitten by a parrot, on several successive nights sprang up out of a sound sleep shrieking, "Take the parrot away! take the parrot away!" I have seen in adults somnambulism perfectly parallel to this. Thus, after a house was robbed, a woman for several nights arose and walked in her sleep, trying to escape from burglars and raise an alarm. The night-terror is, I think, only a form of somnambulism.

Night-terrors which are the outcome of serious brain-disorder are rare, and not to be positively distinguished by their symptoms from those of less serious import. They, however, frequently recur several times a night, and continue for many weeks; whilst the night-terror of irritation usually happens only once, and extremely rarely more than twice, in a single night, and does not continue to recur for weeks, except it be at considerable intervals. Moreover, the serious night-terror is almost invariably accompanied by other manifestations of disorder of the brain-action, which point out its true meaning. Dr. F. Debacker (*Thèse*, Paris, 1881) has reported the case of an infant, who finally died of tubercular meningitis, in whom the earlier symptoms were night-terrors, which were, however, usually associated with spells of fright during the day, and with distinct evidences of hallucination. In the same thesis is recorded an instance in which the nocturnal terrors occurred in a child four years of age and were associated with a rapid loss of power which ended in idiocy; and also a case in which the outcome was epilepsy.

CHAPTER XI.

DISTURBANCES OF INTELLECTION.

For the purpose of studying the symptoms of mental disorder the human intellectual faculties may be separated into the will, the intellectual faculties proper, such as reason, imagination, etc., and the emotions, such as fear, anger, etc.

Disorder of one mental faculty is almost invariably accompanied by a greater or less degree of disturbance of the other mental faculties, but, *a priori*, there seems to be no reason why one faculty of the mind should not suffer alone, and cases are said to occur in practice in which a single faculty appears to be under the influence of disease when no other evidences of mental disorder can be detected.

The human will acts chiefly upon the lower intellectual and emotional brain-functions as a repressive force. It inhibits or puts aside this thought or that distraction or this emotion, rather than brings forward another thought or emotion. We cannot will ourselves into a passion, though we can by a direct effort of the will inhibit or repress a rising anger. If we desire to produce a fit of anger, we do it by bringing before the mind thoughts which act as stimulants to the desired emotion : the almost unconscious recognition of this fact has led to the expression "working one's self into a passion." As is usually the case in disorders of inhibitory nerve-function, affections of the will are most plainly and frequently manifested by weakness or failure of power. The excessive obstinacy and self-assertion seen in certain forms of insanity indicate a condition of abnormal exaltation of the will. Generally, however, extravagances of thought and action which appear to point to an excessive activity of the will are really due to the overpowering action of some emotion or some idea which so dominates the will as to govern entirely the actions of the individual. The obstinacy and self-assertion are under these circumstances really the outcomes of a weakened will rather than of an overpowering egoism,—the person being obstinate or aggressive

because his will is enslaved by a lower intellectual or emotional nerve-centre. Thus, in melancholia inflexible obstinacy may result from the absolute despotism of an overwhelming sorrow. In hysteria the will is probably always abnormally feeble, but the persistence and apparent wilfulness of hysterical subjects are proverbial.

Weakness of the will is produced by various organic brain-diseases, which lower the nutritive tone of the cerebral cortex. It is caused very frequently by chronic poisonings, being one of the most pronounced symptoms of alcoholism and of opiumism. Under these circumstances the subject may show an extraordinary persistency when dominated by his appetite, and yet is really most infirm of purpose, entirely unable to decide upon a course of action in regard to ordinary matters, or to carry out his decision when reached. He is liable to be inordinately influenced by his associates and by his environs, cannot resist entreaty and temptation, and so becomes more and more the sport of his desires and of external influences.

Acute illness, starvation, hardships, age, chronic diseases, any influence which lowers the nutrition of the higher nerve-centres, may produce weakness of the will. So varied are the causes which may produce the so-called *abulia*, or abnormal weakness of the will, that it has no further diagnostic import than to show a serious functional or structural alteration of the cerebral cortex.

Exaggeration of the will-power is known as *hyperbulia*, and shows itself in some forms of mania and of cerebral cortical excitement.

The emotional nature may be by disease depressed, exalted, or perverted; the alteration often affects persistently a single emotion or a single class of emotions, or it may attack successively, at shorter or longer intervals, emotions that are antagonistic. Thus, a subject may be in a continual state of joy or of emotional depression, or he may rapidly or slowly pass from one state of emotional excitement to another, now carried away by anger, now prostrated by fear, now soaring with joy, now overwhelmed by sadness.

In advanced stages of cerebral disease a condition of true emotional enfeeblement or lethargy may be present, so that external circumstances which naturally affect most vividly this or that

emotion fail to produce any response. This mental condition ought logically to be known as emotional depression. It is to be clearly distinguished from excitement or overactivity of the depressive emotions, such as sorrow and their congeners. Viewed in this way, the melancholic person is not in a condition of emotional depression, but in one of emotional excitement,—*i.e.*, of excitement of the depressive emotions. Melancholia is, it is true, frequently associated with depression of the nervous system, but this is not always the case, and the victim of melancholia agitata may be in a condition of general nervous erythrism as pronounced as that which affects the maniac with wildly-expansive delusions. On the other hand, high hopes and abundant joy are in advanced general paralysis closely linked with the most profound evidences of failing nerve-power. If melancholia is to be considered a state of lowered emotional activity, whilst joy and anger are the outcomes of emotional excitement, it logically follows that the antagonistic emotions are different manifestations of one cerebral function, joy being the result of excessive stimulation, sorrow of excessive depression, of the same brain-cells,—a conclusion which I think few persons would be ready to accept as correct.

The relations between the diverse emotions of which I have just spoken are of some importance as explaining the fact that in various mental affections mania and melancholia, or opposite emotional states, may follow each other, and even appear to be produced by the same brain-lesion. Thus, in paretic dementia the persistent hyperæmia of the brain-cortex may cause throughout the attack intense sadness, or an emotional depression may suddenly replace the expansive happiness usual to the affection. To account for such a change it is only necessary to suppose that there is a shifting of the hyperæmia and the excitement from one portion of the brain to another.

The intellectual functions proper may suffer from actual exaltation, giving rise to increase of power; from an exaltation which is so unbalanced as to produce a derangement of action; from a real depression or loss of power.

Absolute increase of mental power is a rare condition, and is never present in any advanced stage of disease. It does, however, sometimes occur. The subject of a pronounced mental exaltation has a passion for intellectual labor, accompanied by a corre-

sponding power of accomplishment. It is no longer an effort to fix the attention upon an intricate subject for successive hours. The sense of fatigue is lost, and the brain works on without pain, the quality as well as the quantity of the result being beyond that which the individual in his normal condition can produce. This state of mental exhilaration sometimes comes on during protracted mental labor. It is probably always associated with hyperæmia of the brain-cortex, and is usually accompanied by pronounced insomnia. It is a very dangerous condition, and should be the signal for immediate cessation of mental effort and for medical treatment. It is sometimes developed without obvious cause as a prodrome of severe mental disease. Thus, I have seen it precede a fatal outbreak of acute phrenitis, and it may usher in paretic dementia.

If one or more of the mental functions are excited entirely beyond the control of the will, and judgment becomes impossible, a mental condition is produced which in its most severe acute form is sometimes spoken of as delirium, and in its milder or more chronic forms as insanity.

Failure of the mental powers is a very common result of functional and organic brain-disease. When complete it constitutes the condition known as dementia.

It is often of vital importance to recognize the dawnings of mental failure. The failure usually manifests itself first in loss of memory. This has already been sufficiently discussed (see page 369). Next to memory in the order of implication, and sometimes even preceding it, is the power of fixing the attention. The mind of man naturally wanders from subject to subject. A continuous thoughtful application depends upon the exertion of the inhibitive power of the will in repressing distracting thoughts and shutting out new perceptions. The power of persistent attention to one subject is to a great extent acquired by training. Its exercise is a large feature in all severe intellectual work. Consequently, when the brain is exhausted not only do the reasoning faculties labor with difficulty, but increased effort is required from the weakened will to maintain the necessary fixity of attention. Mental toil becomes, therefore, most irksome, as is recognized by the common expression of sufferers that "work is becoming more and more of an effort." Failure of memory and failure of

the power of fixing the attention have no particular diagnostic import. When they coexist and are associated with any other evidences of mental derangement, they indicate a serious disease of the brain itself. The loss of the power of fixing the attention, however, when it exists alone, usually depends upon simple cerebral asthenia,—a condition in which there may also be some loss of memory.

A symptom which may depend upon either mental excitement or loss of mental power is *incoherence*. An incoherence due to a heightened but irregular cerebral activity results from the excessive rapidity of the intellectual acts, as well as from their disconnected sequences. Before one idea is fully translated into words, another rushes into expression, and a hopeless confusion of talk results. The ideas tumble out as it were over one another. Incoherence from lack of mental power, on the other hand, arises either from the inability to complete the mental act or from the lack of the power of translating it into suitable words. In typical cases there is little difficulty in distinguishing between these varieties, which it is allowable to call respectively *active* and *passive* incoherence. The rapid utterances of the raving maniac usually show most plainly that his mind is pouring out broken hints of an infinite series of jostling ideas; whilst the slow, confused, disconnected, hesitating words of the demented no less unmistakably portray his inability fully to conceive an idea and embody it in words. There are, however, cases of disease in which mental excitement coexists with failing power, and in which, therefore, the incoherence is of mixed type.

Human character is the result of the established balance between the will, the intellectual attributes, and the emotional forces of the individual. When any of the correlated factors are altered there must be a corresponding change in character. Character is, therefore, always seriously implicated in mental affections. Not rarely changes in the intellectual or emotional nature so subtile or hidden as not to be readily perceived register themselves with astounding distinctness on the dial-plate of character. Hence alterations of character are of the weightiest diagnostic import. They may be the first evidences of a developing pure insanity, but when sudden and severe they usually point towards dementia paralytica. A primary sudden criminal outbreak in dementia

paralytica is generally sexual in its direction. Thus, in a case now under my care the first marked disorderly action was an attempt to rape a servant-girl. After this it was discovered that very large and foolish purchases had been made as the beginning of a grand business scheme entirely foreign to the daily occupation of the man. An estimable citizen goes to a distant city and attempts to turn a hotel into a bawdy-house. Another, whilst still performing acceptably the duties of an important public office, tries to seduce, and, this failing, to rape, his own daughter.

In dementia paralytica, as in the pure insanities, the moral degradation may, however, run in other than sexual channels. The temperate man suddenly becomes addicted to drink; the honest man all at once appropriates large sums of money, which, it may be, he spends in licentious revels; he who has always been exceptionally self-controlled becomes violently passionate; the amiable, loving husband and father changes into a household demon. Careful examination under these circumstances will usually detect other symptoms of paretic dementia. The evidences to be searched for are failure of memory, deterioration of mental faculties, inequalities of the pupil, perceptible loss of physical endurance or of the power of doing fine, complex physical acts, habitual emotional states of *bien-être*, and a tendency to expansive delusions, as shown in the subject's estimate of his own powers, business prospects, or schemes, and of the value of his possessions or surroundings. Whenever any of these things can be found, it is the physician's duty to give warning to the friends of the patient, and, with their assent, to act. There are certain specific symptoms whose relation to cerebral diseases is so close and so important that they demand very careful consideration. These symptoms are naturally divided into two sets: first, those which indicate disorder of the perceptive faculties; secondly, those which are connected chiefly with the intellectual and emotional spheres. Under the first head I shall consider Hallucinations and Illusions; under the second head, Delusions, Imperative Conceptions, Morbid Impulses, and Morbid Desires.

An *Hallucination* is the perception by any of the senses of an object which has no existence. It is the conscious recognition of a sensation of sight, hearing, feeling, taste, or smell which is not due to any impulse received by the perceptive appa-

ratus from without, but arises within the perceptive apparatus itself: in other words, an hallucination is a subjective sensation which assumes the definite attributes of an objective sensation. It is commonly simple,—*i.e.*, connected with a single sense. Thus, the vision is usually seen, not seen and felt. The false voice is heard, the mysterious presence is felt, but the presence and the voice usually do not coexist. In the order of their frequency of implication the senses may be enumerated as follows: sight, hearing, touch, smell, taste. The particular characters of the perceived object vary indefinitely, and involve the whole range of perceptions. Every variety of color and form, of sound and odor, of feeling and taste, may be perceived.

In some cases, as in mirage, a *false perception* may amount almost to an hallucination; that is, an impulse from without may give rise to such a distorted misleading conscious perception that the person really sees or feels or hears that which has no existence. A distorted sensation, or, in other words, the perception of an object in characters which it does not possess, is frequently spoken of as an *illusion*. In nature there is no sharp line between illusions and slight distortions of the perception of objects, or between illusions and hallucinations. An hallucination may be caused by an external stimulus so slight that it cannot be discovered, but it may arise entirely from within the nervous system.

An hallucination has no definite diagnostic import. It may come from exhaustion of the nervous system, especially when there is at the same time an intense desire. Thus, the wife, worn out with long watching and grief, sees in obedience to her yearnings the living form of her dead husband. The monk, exhausted by long prayer and fasting, if consumed by ardent devotion, is visited by saints or angels, or, if he be tormented by suppressed sexual desires, is haunted by troops of tempting devils or voluptuous sirens. The person perishing with thirst sees or hears cool springs, babbling brooks, or plashing fountains; gorgeous feasts float before the vision of the starving, and the shipwrecked mariner is tantalized by rescuing barks.

Hallucinations may be the result of the immediate action of a poison, as in the beatific visions of the hashcesh-eater, or may be the outcome of the peculiar nervous state which follows the abuse of narcotic stimuli, as in delirium tremens. Conditions of

the nervous centres at present inexplicable may call hallucinations into being, as in hysteria. More rarely the hallucination is the result of an organic brain-disease, when its nature is almost invariably pointed out by coexisting symptoms, such as epileptic paroxysms or local palsy. The structural alteration in such cases is commonly in the nerve-tract especially connected with the affected sense.

An hallucination does not depend upon or prove the existence of intellectual unsoundness. It is, however, very apt to be associated with such unsoundness, because the condition of the sensory brain-tract which produces it is apt to accompany a similar condition of the higher or intellectual centres. Moreover, it often affords us a means of testing the condition of the brain-centres. If the judgment fails to correct the testimony of the disordered sense by that derived from other senses, the subject is of unsound mind. When, for example, the individual believes that the vision that he sees or the voice that he hears really exists, then is his judgment dethroned. It will be readily seen that in such a case it is not the seeing of the vision, but the loss of the power of weighing evidence, that is the proof of the intellectual degradation. As will become very apparent during the discussion of delusions, the hallucination, in the case just imagined, has given rise to a delusion.

The word *delusion* may be defined to be a false belief, but as it is used by alienists the term means something more than this. By Spitzka the insane delusion is said to be "a faulty belief out of which the subject cannot be reasoned by adequate methods for the time being." The objection to this definition is that there are many faulty or false beliefs held by perfectly sane persons out of which such persons cannot be reasoned, but which are not insane delusions. Thus, either the Christian or the Mussulman, under such definition, is the victim of an insane delusion. To meet the necessities of the case the definition should be modified so as to read, "A faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being."

The parallelism between a delusion and an hallucination is very close. A delusion is a false belief. An hallucination is a false perception. The delusion becomes an insane one only when the

false belief cannot be dissipated by absolute proof of its incorrectness. The hallucination becomes an insane one only when the false perception cannot be corrected by the judgment through the other senses. In either case the essence of the insane mental state is loss of power to receive and weigh adequate evidence.

Thus, John Smith hears voices where there are none; he is insane only when he is unable to correct the evidence received through the sense of hearing by that received through the senses of sight and feeling. If he persistently believes that persons speak to him, although he cannot see or touch them, his judgment is in abeyance. On the other hand, John Jones believes that a certain barn exists upon a certain field where there is no barn. Under these circumstances he has a delusion, a belief which has grown up in his mind from some cause unknown. Now, if, when taken to the field, he is incapable of receiving the evidence of his senses and persists in his belief that the barn is there, he is insane; but if he receives the evidence of his senses and perceives that the barn does not exist, he is not insane. In case of insane hallucinations or delusions, the truth or falsity of the vision or of the belief is not essential. The essential thing is the condition of the mind of the individual,—a condition which prevents it from receiving evidence. Hence an insane belief may be true although insanely held.

In the supposititious case given above, assuredly the mental state of the individual is in no wise dependent upon the absence of the barn, although such absence renders a test of the subject's mental condition possible. The distinction just drawn may seem unimportant and so trite as to be unworthy of discussion, but the failure to understand it has been one cause, in my experience, of the inability on the part of learned lawyers to comprehend the subject of insanity.

Not long ago, after due process of law, an insane man by the name of Taylor was hung in Philadelphia for the unprovoked murder of a prison warden. It was in evidence that the man believed that all the attendants of the prison were Catholics and were "down on" him because he was a Protestant, and were destroying him. The prosecuting attorney asked, "Supposing it were proved that the prison attendants were Catholics, would it not have to be acknowledged that the man's belief was correct, and

that he was not insane?" Apparently neither lawyer nor judge could be made to understand that the falsity or the truth of the prisoner's belief in the Catholicism of the attendants had little to do with the question of his insanity. It was proved that he had other delusions of persecution, and his having adopted a belief in regard to the Catholicism of his attendants which was in accord with such delusions, without any evidence of their alleged Catholicism, and having reasoned insanely upon the subject and acted in accordance with conclusions so reached, showed that his action rested upon mental unsoundness. Surely the "*because I am a Protestant*, therefore they were destroying me," ought to have made the mental condition of the prisoner clear. In the language of Spitzka, "Repeatedly does it occur in the alienist's experience that the facts of a case and the delusion *happen* to correspond." This is well illustrated in a case reported by him. An artist's model asserted that he was the finest-built man in the United States. He really had a magnificent figure, but his announcement was, notwithstanding, that of a paretic dement, for inquiry elicited the statement that the "girls looked at him because he had a peculiar expression in his eyes which they fancied," and he revealed other unmistakable evidence of general paralysis.

An insane belief or delusion may rest upon an hallucination, may be built upon a foundation of disordered sensation, may spring from the most trivial circumstance, or may, so far as can be judged, be self-engendered in the mind. Thus, the voice that is heard as an hallucination gives rise to the delusion of an ever-present persecutor; a persistent distress in the abdomen, to a delusion of pregnancy, or that the bowels are dropping out, etc. The following case from my note-book illustrates very forcibly the curious way in which a delusion develops in the mind without the slightest foundation in verity. A man after a malarial fever began to have suspicions in regard to the chastity of his wife. For a time he kept these to himself, but finally he accused her of infidelity. After this had continued for some weeks he presented himself with his wife at my clinic, saying to me, "I think my wife goes with other men. She thinks I am crazy. I am uncertain whether she or I am right." On being questioned, he stated that he first noticed her looking behind her, as though she

were looking for some one, when they walked together; that he afterwards saw a handkerchief lying on the bureau in her room, just as she would have left it if she had been flirting with some one out of the window, and that when he saw a chair by the window of her room and a man at the corner of the street he was convinced that his suspicions were correct; in this he was corroborated by finding three dollars in a trunk, which he believed his wife had received "for evil courses," although she had declared that he himself had given it to her. He further stated that he watched her eyes. In a very eager, tremulous manner he said, "I got a lamp, and when I found her eyes were dark beneath I told her there was something wrong with her, and then she began to think there was something wrong with me. I firmly believed she was going with other men." The man had an inherited tendency towards insanity, and had lost much sleep. When his whole case was thoroughly explained to him, he said that he "now understood it, and was glad to hear it, and that it gave him power to brace himself against the notion," ending with the assertion that he believed that "he had a good woman." In reply to a question, he said, "I do not think there is danger of my hurting my wife, but these things come on me so that I cannot control myself at times, and I am willing to go to an asylum if it is thought to be right."

The relation between the emotional state of an insane man and his delusions is very close. Expansive or happy delusions accompany emotional exaltation, while horrible or sorrowful delusions go hand in hand with depressive emotions. Thus, the melancholic woman is oppressed with the belief that she is hopelessly damned, that her husband is unfaithful, or that she is pregnant with devils; whilst the maniac, overflowing with animal spirits, is a prophet sent of God, is owner of uncounted millions, or mayhap is about to become the mother of the Messiah. The emotional state and the delusions constantly react upon one another. Some alienists believe that the character of the delusion is directly dependent upon the dominant emotion; but it seems to me more probable that the characters of the emotions and of the delusions are the result of a common cause, rather than that either governs the other.

The nature of delusions varies so indefinitely as to render any

attempt at a thorough classification futile. There are, however, certain classes of delusions which are so frequently met with and so characteristic as to require especial study. The most important of these are—1. Expansive Delusions. 2. Hypochondriacal Delusions. 3. Delusions of Persecution.

Expansive Delusions usually concern the personality of the individual who has them, either as to his prowess, his mental or physical attainments, his possessions, or his future prospects. The patient boasts that he is the strongest man in the world, asserts that his mental powers are immense, or that he is a king or other notability, or more commonly talks of his millions of money, his gold-mines, his farms of unlimited extent, his vast stables full of unnumbered horses of the choicest breeds, his far-reaching and gigantic business schemes, etc. This condition constitutes the *délire de grandeur*, and, whilst in the majority of cases it depends upon the existence of general paralysis, it may be present in many forms of mental disease. I have seen it very pronounced in cerebral syphilis, and have watched the millions of dollars possessed by the subject shrink to thousands, and the thousands to hundreds, as the brain-lesions grew less under the administration of mercury. Then even the hundreds disappeared, and his own poverty was confessed; but the assertion still remained that "his uncle was worth a million," until at last this too vanished in the recognition of the desolate truth.

Hypochondriacal Delusions relate to disease of the person of the patient, and are usually, but not always, associated with a depressive emotional state. They sometimes rest upon a substratum of ill feeling, or even of actual disease, in the part alleged to be hopelessly affected. They are often obviously absurd, as that the legs are made of glass. Of all forms of delusion this is the one in which the gradations between the sane and the insane belief are most subtile. Every step can be found between the slightest exaggeration of symptoms and the hypochondriac foundationless belief. Unless a hypoehondriacal delusion is upon its face absurd, the physician must be very careful in basing upon it an opinion that the subject of it is irresponsible, since many invalids are hypochondriacs and have exaggerated beliefs bordering closely upon delusions, but are, nevertheless, of sufficiently sound mind for the performance of the ordinary duties of life.

Delusions of Persecution are common in melancholia, but are not always associated with a pronounced depressive emotional condition. They are always the source of great annoyance and distress to the subject, and are usually associated with hallucinations, which I think are most apt to be connected with the sense of hearing. Very commonly obscene, reproachful, or threatening voices are heard at all times and in all places. Usually the delusion of persecution does not attach itself in the mind of its victim to one person, but to classes of people or to unseen spirits. Sometimes, however, the delusion does affix itself to one individual, as in a recent case in which a woman travelled across the continent of America to kill a doctor who she believed was placing a spell upon her. Of all the quiet classes of the insane, those who have delusions of persecution are the most dangerous. They are impelled by motives of revenge and of fear to kill those who are persecuting them. This is especially the case when the delusion attaches itself to one individual; but even voices in the air may lead to sudden violent assaults upon by-standers who are for the moment thought to be the source of the words. Moreover, the lunatic may at any time fix in his mind upon any acquaintance or notable person as the origin of his persecution and make his plans in accordance.

A very important division of delusions is into systematized and unsystematized. A *systematized delusion* is one concerning which the subject reasons, and which he defends more or less logically. Any character of delusion may be systematized. If a lunatic asserts that he is worth a million of dollars and simply sticks to his belief when it is denied, he has an unsystematized delusion of grandeur; but if he should attempt to defend his delusion by describing how he had inherited his wealth or how he had acquired it through investments or business ventures, his delusion would be systematized. Again, a person suffering from melancholia believes that his soul is lost. If, when opposed, he simply reavows his belief and assigns no reasons for it, his delusion is unsystematized; but if he says he is lost because he has committed the unpardonable sin, quotes Scripture to show that such a sin warrants his doom, and perhaps tells why and when he sinned, his delusion is systematized.

Great diagnostic value has been attached by some recent writers

to the distinction between systematized and unsystematized delusions, and much has been predicated upon it in the classification of insanities. According to my experience, however, in nature every gradation is to be found between the most thoroughly systematized delusion and that which is most completely isolated. I have seen various cases in which it was doubtful whether the delusion should be classed as systematized or unsystematized; and, whilst I acknowledge that in typical partial insanities the delusions are systematized and in typical general insanities they are unsystematized, I am of the opinion that in this character, as in others, the two groups of general and partial insanities pass in nature insensibly into each other.

There are certain conceptions or general ideas which may arise in the brain of a person, and to a greater or less degree dominate his actions, although the reason may not be unsettled, and the falsity of the conception may be recognized by the individual whom it controls. Such a phenomenon is known as an *Imperative Conception*, and differs from a delusion in that its falsity is recognized, although the individual is powerless to withstand its influence. Closely allied to the imperative conception is the *Morbid Impulse*. Some alienists, indeed, teach that the imperative conception gives rise to the morbid impulse. In certain cases this undoubtedly happens, as when the imperative conception of personal defilement gives origin to the impulse of escaping from that which defiles; but a morbid impulse may arise without any discoverable imperative conception. Thus, I long had under my care a man in whose family insanity was distinctly hereditary, but in whom the only symptom that I could find was an impulse to assault bystanders,—an impulse apparently born of no reason, although felt with such urgency as to fill the patient with a terror of himself. Once, upon returning home, I found this man sitting in my office terribly excited, and greeting me with, "Doctor, doctor, I nearly did it! I nearly did it!" It appeared that he had spent forty-eight hours without intermission in a vortex of political excitement, and suddenly the impulse to kill had come on him with such power that only by fleeing to my office was he able to save himself. The impulse to throw one's self from a precipice, caused by standing on its brink, is a familiar instance of a mild morbid impulse without an apparent foundation of an imperative con-

ception; whilst the reasonless dread which many persons have of a snake, toad, cockroach, or other harmless creature probably depends upon an incipient imperative conception of personal defilement.

The act which results from a morbid impulse is sometimes spoken of as an *Imperative Act.* An imperative conception is viewed by some alienists as an "undeveloped delusion." It is, however, not a proof of general mental unsoundness, but in some cases finally the reason of the patient fails to recognize the untruthfulness of the imperative conception, which conception thereby becomes converted into a delusion, precisely as an hallucination may give rise to a delusion.

A very important and common imperative conception is a morbid fear. This may take almost any form, and may be simply an exaggeration of a normal feeling or may arise *de novo.* Thus, in some persons the fear of a thunder-storm is so violent as to destroy for the time being all rationality; in others the natural dislike of filth is increased until it dominates every action of life. On the other hand, the horror of walking in an open place, which is sometimes so overwhelming, seems scarcely to be based upon any natural feeling. To many of these morbid fears names have been given by systematic writers. The fears, however, vary so in their detail that it is not possible to express them accurately and fully by any system of nomenclature. A few of these names may be cited, as representing the more characteristic forms of morbid fear. The following list, taken from Dr. Beard, portrays very well the absurdities of nomenclature:

Astraphobia, fear of lightning. Topophobia, fear of places,—a generic term, with these subdivisions: Agoraphobia, fear of open places; Claustrophobia, fear of narrow, closed places. Anthrophobia, fear of man,—a generic term, including fear of society. Gynæphobia, fear of woman. Monophobia, fear of being alone. Pathophobia, fear of disease,—usually called hypochondriasis. Pantaphobia, fear of everything. Phobophobia, fear of being afraid. Mysophobia, fear of contamination.

As illustrating imperative conceptions, a few cases from my own experience may be cited. A very strong shoemaker, past middle life, was oppressed with the idea that he could not walk unless he had some covering over his head. On a stormy day

the natural cloud-canopy sufficed, and on a clear day an umbrella carried over his head gave a measure of relief, so that he was able to command his movements. He could walk in a thick wood, but, as he himself said, if ten feet of clear sky intervened between the wood and a spring he would die of thirst before he could cross over. No other symptom of physical or mental ailment could be detected.

A lady had a dread of personal defilement: hundreds of times daily she washed her hands, without avail; bank-notes fresh from the press were the only money she would use; a door-knob she would never touch, but would remain in the room until some one opened the door; in putting on her clothes only the inside of each piece was touched by her fingers, and this as daintily as possible. Without entering into further details, suffice it to state that her whole life was arranged in order to avoid as much as possible contact with any person or thing. On my asking her to shake hands her embarrassment was extreme: though naturally polite, and feeling under some obligation to me, she was nevertheless entirely dominated by her imperative conception. Finally she said, "Dear doctor, don't ask me: you know you touch so many people."

A gentleman entirely rational, able to manage his business affairs well and to converse on all subjects, was completely ruled by imperative conceptions and morbid impulses, the connection and the independence of which are well illustrated by his case. Thus, for many years he had an impulse continually to rub his arms against his sides, and this he did incessantly until coat after coat was rubbed into holes. No morbid conception could be found underlying this or some of the other impulses which he had. Nevertheless, he did have imperative conceptions with outgrowing secondary impulses. For many months he was markedly mysophobic. Then he had the conception that he must lay things down straight and could not do it. Most of his waking moments were at this time spent in putting down and arranging. When he placed a book on the table, over and over and over again he would lift it up, straighten it, pick it up and re-lay it, etc. Often at night he would be two or three hours getting away from his coat, which he was perpetually arranging upon the chair on which he had laid it. There was no delusion, and on my asking

the man why he yielded to the impulse, he said, "I can resist it for a while, but after a time the same overpowering sensation comes as when I hold my breath, and I must do it. I have found that if I say very fast, 'It is straight, it is straight,' over and over again, at the same time craeking my fingers briskly by shaking my hand, the impulse often suddenly vanishes, with immediate relief."

The relation of imperative conceptions and morbid impulses to insanity is a matter of great theoretical and practical interest. They are undoubtedly frequent in the insane, and usually careful examination of a case in which they are present will reveal distinct symptoms of alienation. They may, however, exist in persons whose intellectual actions are in other respects entirely normal, and in whom the judgment is not dominated by the conception which may influence the actions against the judgment. To himself the sane subject of an imperative conception seems possessed by a demon whom he must obey.

The relation of morbid conceptions and impulses to legal responsibility for acts committed involves questions of great practical difficulty. The victim of the morbid impulse cannot properly urge such impulses as excuses unless the deed in question is immediately produced by them. When the act is committed because the actor is forced to do it by a morbid impulse, the actor is, of course, morally blameless; but who can tell whether the impulse was resisted to the uttermost? Moreover, the needs of society, and the case with which such impulses could be alleged or counterfeited, very properly give us pause in attempting by them to excuse a criminal act. The clearest possible proof should be required that the impulse was really morbid and irresistible.

Names have been given to various morbid impulses. In most cases these names are misleading in their etymology and primary meaning. They usually end in "mania;" but the morbid impulse is not a mania, but a symptom which may either coexist with maniacal manifestations or be isolated. Thus, *pyromania* is a morbid impulse to set fire to buildings; *kleptomania*, a morbid impulse to steal; *homicidal mania*, a morbid impulse to kill; *suicidal mania*, a morbid impulse to commit suicide, etc. Unfortunately, the nomenclature is made still more complicated by the fact that often when the morbid impulse exists in an insanity the name

usually applied to the impulse is given to the whole attack. Thus, a melancholia with an impulse to set fire to houses would be called pyromania. Not rarely, indeed, there is not even the excuse of the existence of a morbid impulse for the name given to the disease. Thus, the man who, not believing in a future existence, commits suicide because he is suffering from the unutterable misery of melancholia, is logical and reasonable in his suicide, and does not kill himself through any *morbid—i.e.*, unreasoning—impulse. Suicidal and homicidal maniacs are simply persons who have a tendency to kill themselves or others.

Morbid desires are exaggerations or perversions of natural appetites, and are chiefly seen in regard to hunger and the sexual passion. Mere depravity and wickedness may convert man into a monster: neither cannibalism nor the lowest sexual degradation is necessarily the offspring of disease. Nevertheless, disease may affect the appetite for food or for sexual congress as it does other functions of the nervous system.

In mania, in paretic dementia, in hysteria, indeed, in almost any form of insanity with excitement and exaltation, the sexual passion may become an all-devouring, insatiable lust. In the female this condition is known as *nymphomania;* in the male, as *satyriasis.* The victim of it talks incessantly and indecently about sexual congress, makes furious love to all persons of the opposite sex, exposes the person, etc. *Erotomania* is a very frequent condition, in which there is the appearance but not the reality of sexual excitement. The subject of it conceives a strong attachment for some person of the opposite sex whom perhaps he or she has never seen, and lives in a perpetual worship. Sometimes the object is in public life, and is followed from place to place with a pertinacity and publicity which may amount to actual persecution. Even if opportunity offer, the erotomaniac makes no effort at cohabitation. Satyriasis leads to sexual excess and to rape. Erotomania is a platonic affection, which involves the higher conceptive sphere rather than the lower nerve-centres, and leads to sexual abstinence.

The individual symptoms or manifestations of disordered mental action having been sufficiently discussed, the consideration of the so-called mental diseases is in order. Before, however, entering upon the subject of insanity it is necessary to discuss the pro-

found active disorder of intellection connected with constitutional affections to which the name delirium is given.

By the term *delirium* is meant an acute mental condition in which there is incessant, more or less incoherent talk, which is not directly inspired by surrounding objects,—the sufferer being so occupied with his own mental conceptions that he is not entirely conscious of his situation: indeed, in most cases there is not a true consciousness. Delirium may be either low in type, or wild and furious. It is produced by a large number of diseases which are not immediately connected with the nervous system: under these circumstances the cause of the mental aberration is to be made out by diagnosing the disease which produces it. It is a general law, with few exceptions, that a delirium which is low and muttering, if acute, and not preceded by protracted evidences of cerebral disease, is due to some affection not immediately connected with the brain. Violent acute delirium is often the result of brain-disease, but may be secondary. In pneumonia occurring in persons exhausted by dissipation a wild delirium may be the most prominent symptom, and give rise to the false diagnosis of phrenitis. In every case of sudden severe delirium the lungs should be carefully examined, when the physical signs may demonstrate a pneumonia, although there may be neither cough, pulmonic distress, nor apparent disturbance of the respirations.

A sudden severe delirium may mark also the onset of an acute general disease, such as malarial fever, scarlet fever, etc. Usually in such a case the delirium itself is low and muttering; but, even if it be fierce, the pulse is weak and feeble, the countenance is depressed, and a general expression of vital failure exists, which to the experienced eye at once indicates the presence of a depressing poison in the blood.

Before entering upon the discussion of the classifications of insanity, the question how much of abnormal mental action is compatible with sanity seems naturally to present itself. Its answer involves the definition of the words sanity and insanity, and, like these definitions, probably will always be unsatisfactory. Insanity is not a definite disease, but an abnormal state, varying indefinitely in its intensity,—separated by no tangible line from sanity,—arising from a number of diverse diseases, and termi-

nating in most various ways. It is a mental weakness; and it would be as absurd to ask for a definite line separating the physically weak from the physically strong as to ask for one separating the mentally weak from the mentally strong.

For his own purposes of science, or even of treatment, the physician needs no definition of insanity, but the relations of man to man are so altered by insanity that the law must take particular notice of the subject of insanity. Even, however, for the purposes of the law insanity is not a fixed term, because it is a well-assured axiom that a man may be legally sane—*i.e.*, responsible—for one class of acts, and insane—*i.e.*, irresponsible—for another class of acts.

As already contended, there can be no scientific definition of insanity except that it is a state of mental aberration. Such a definition does not meet the needs of the court-room, which demand an arbitrary although shifting line between the sane and the insane. The term insanity as used by judges and lawyers is legal rather than scientific, and the law ought clearly to define the word. It does, however, no such thing. It does not frame an authoritative definition of insanity, but through the mouths of its exponents puts forth an abundance of contradiction.

Probably as good a definition of insanity as the expert can frame to meet the clamor of lawyers is, that insanity is a condition of mental aberration sufficiently intense to overthrow the normal relations of the individual to his own thoughts and acts, so that he is no longer able to control them through the will. The difficulty of applying this definition to the individual case consists in the fact that the will does not all at once lose its grasp on the lower faculties, but that little by little these slip from under its control. Of degrees of responsibility none but the All-knowing can judge, and to say with assured correctness just when the lost control has been lost is not given to mortals. In a court of justice it becomes the expert to state as nearly as may be the exact mental condition of the prisoner, leaving to the judge the decision as to the legal responsibility of the prisoner,—*i.e.*, the relation of his mental condition to the law of the commonwealth in which the trial is held.

Insanity being a symptomatic condition, and not a disease, it is

illogical to consider different forms of it as distinct diseases: the best that can be done is to describe the diseases of the brain and the insanities which accompany them so far as we know such diseases, and, when our knowledge of diseases fails, to describe forms of insanity not as diseases but as symptom-groups.

The purposes of discussion necessitate the naming of these symptom-groups, for it becomes essential to have short terms which shall convey a whole group of symptoms at once to the mind. Naming symptom-groups naturally leads to the delusion that these groups are diseases: hence melancholia, mania, etc., are constantly written about as though they were terms of equivalent force to typhoid fever or scarlatina, whereas they are simply of the same rank as diarrhœa or paralysis.

This is shown by the following facts:

1st. Similar mental symptoms may be produced by various organic brain-diseases; or, as Dr. Charles F. Folsom says (*American System of Practical Medicine*, vol. v. p. 202), "tumors, new growths of all kinds, exostoses, spicules or portions of depressed bone, embolisms, hemorrhages, wounds, injuries, cysticerci, may give rise to any of the symptoms of the various psycho-neuroses and cerebro-psychoses."

2d. Almost any form of insanity may exist without demonstrable organic lesion. This is shown by the well-known fact that in a large number of autopsies upon the insane skilled observers have failed to detect alteration of brain-structure.

3d. Antagonistic forms of insanity may be produced by lesions which are, so far as we can perceive, identical: as is witnessed by the circumstance that in paretic dementia the usual expansive delusions may be replaced by a profound melancholy. Further, lesions usually accompanied by insanity may exist without mental disorder. Dr. Folsom says, "Indeed, nearly every pathological condition of the brain known in insanity—in kind, if not in extent and degree—may be found in diseased or injured brains where there has been no mental disease in consequence."

4th. The form of the insanity may change in the individual without appreciable cause and without conceivable change of disease.

5th. Almost every grade of case exists in nature, uniting by an unbroken series the various insane-symptom groups. Thus,

of the two most antagonistic forms of acute insanity, acute mania and acute melancholia, Bucknill and Tuke say (Phila. edition, 1874, p. 427), "Between acute mania and acute melancholia no distinct line of demarcation can be drawn. The domains of the two diseases overlap so much that, in practice, cases not infrequently present themselves which may with equal propriety be referred to one or the other."

The considerations which have been brought forward show that the various forms of insanity are not entitled to be considered as distinct diseases, and that at present we cannot connect cerebral lesion and mental symptoms in their causal relations. More than this, the rapid recoveries which sometimes occur in apparently hopeless cases of insanity show that the symptoms cannot depend upon alterations of the brain-substance sufficiently gross to be detected by our present methods.

I shall narrate, as showing this, a single case, that of a lady with whom I was thrown in almost daily contact for many years. At about the age of forty-five she was taken with religious melancholia of the most pronounced character, which was accompanied by agitation, and sometimes by frenzy. This persisted for fifteen years. There had been in all this time not the slightest wavering of the mind of the woman in regard to her future life. She firmly believed that her soul was irretrievably lost. At the same time her general emotional nature had undergone a retrograde change: she had become exceedingly jealous of attentions paid to other persons, and had lost many of the peculiar traits of refinement which had been her especial characteristic. After being in an asylum for some time, she recovered intellectual power sufficient to enable her to take charge nominally of her husband's house, which was really managed by her attendant, but there was no wavering in her delusion, nor even any temporary abatement of her misery.

One night the attendant noticed this lady on her knees at the bedside. This was the first time in fifteen years that she had been known to kneel in prayer. The nurse, being a wise woman, did not disturb her, and there she remained all night. In the morning she joined the family, and said that she had found Christ, and that she was perfectly well and happy. Her old disposition had returned, and her peculiar jealous sensi-

tiveness had disappeared. The woman who had been buried for fifteen years had emerged in one night without even the grave-clothes about her. This continued for one week. Then the old cloud came on her, and for days she was in the old condition; but suddenly the sunlight again broke through the clouds, and she remained well for three or four days, to relapse, and after some hours again to regain her sanity. These attacks continued to recur at gradually lengthening intervals. Finally she had been perfectly sane for several consecutive months, when suddenly she was seized with a serous diarrhœa, causeless as far as could be ascertained, and hopeless as far as relief by remedies was concerned. In forty-eight hours she was dead. I believe that the cause of that death was the same obscure something which had so potently affected for years the emotional life: that which for so many years had dominated the nerve-centres of higher life attacked and paralyzed the lower centres of animal life, and death came speedily.

We can scarcely conceive the nature of a lesion which, after having held for fifteen years the nerve-centres in an iron grip, suddenly let go its hold. For its demonstration the microscope is useless. Our best instruments show us in human spermatozoa nothing but irregular, transparent specks of protoplasm, not to be distinguished one from the other. Yet the records of past generations are written in the little formless particles, in which also are enfolded the potentialities of future successions of men. Structure and function seem so widely independent that it is almost hopeless to expect that we shall ever understand the infinitely delicate changes which take place in the complex protoplasm of the brain, and to be able to say why waves of emotional and mental paralysis sweep over the individual. I believe that the changes are physical, but I also believe that it will never be within human power to recognize their nature. The microscope is a coarse, blundering tool, powerless to reveal the ultimate changes of nervous protoplasm gone mad.

Almost every systematic writer upon the subject has his own private classification of insanity,—a fact which is strong evidence that no classification as yet made, or as yet possible to be made, is scientifically accurate. Much of the confusion arises out of the false view that the so-called distinct insanities are distinct diseases.

If it were once generally acknowledged that almost all of these forms of insanity in nature shade into one another, and that the separations are arbitrary, simply made for convenience of study and discussion, the simplest arrangement would become popular because the most convenient. The following arrangement is more simple than novel, and better adapted to the need of the practical alienist and student than to that of the theorizer.

Group I.—Complicating insanities, in which there are distinct physical symptoms of disease of the brain, the cerebral disorder not being due to an acquired or inherited constitutional diathesis.

Group II.—Constitutional insanities, in which the cerebral disorder is due to an acquired or inherited constitutional vice, including in the latter term diathesis, constitutional diseases, and subacute and chronic poisonings involving wide-spread areas of the body.

Group III.—Pure insanities, in which the mental disorder is not accompanied by essential symptoms of organic brain-disease or dependent upon a diathesis.

COMPLICATING INSANITIES.

Almost any form of organic brain-disease, such as abscess or tumor, may be accompanied by mental disorder. If the gross lesion be focal, it gives rise to focal symptoms, by which its existence is betrayed. In other words, the character of a focal lesion in complicating insanities is to be made out by a study of the purely physical symptoms, it being borne in mind that profound mental aberration of a chronic type, and not accompanied by stupor, indicates a wide-spread cortical lesion rather than a focal disease. The cortical lesion may, however, be secondary to a focal disease.

The only wide-spread brain-diseases which produce Complicating Insanities are Meningitis, acute and chronic, and Periencephalitis, acute and chronic.

Meningitis.—Acute meningitis is very rare in the adult, but may be the result of a sunstroke or of a traumatism. It is usually secondary to a chronic meningitis, a brain-abscess, or other organic cerebral affections. If, as is very rarely the case, the chronic disease has been obscured and perhaps altogether over-

looked, a secondary meningitis may appear to be a primary affection. This I have especially seen in cerebral syphilis, when the original gummatous tumor has probably been very limited in its extent. It is probable that an acute meningitis may be produced directly by septic poisoning.

The symptoms of an acute meningitis are furious delirium, with wild outcries, great restlessness, perpetual fighting, and often convulsions, the attack being preceded by an agonizing headache, which persists as long as consciousness is retained. There is always in the beginning pronounced fever and excitement of the circulation, which is revealed by a rapid, bounding pulse, or by one which is small and very hard,—*i. e.*, the "corded pulse." The disease is often ushered in by a rigor, vomiting is frequent, and violent epileptiform convulsions may mark the abrupt onset. The headache and mental excitement are intensified by bright lights or loud sounds. The convulsions may be partial, and in mild cases the motor disturbance may be manifested by persistent muscular rigidity, which, when the base of the brain is chiefly affected, is most marked in the neck. The stage of excitement lasts from a few hours to several days, and is followed by one of paralysis and depression, in which there is stupor deepening into coma, a slow, intermittent pulse, or other evidence of failing circulation, and finally death amidst wild convulsions, or, it may be, in profound muscular relaxation.

The meningitis of which I have so far been speaking is the sthenic disease as it occurs during adult life. In childhood inflammation of the brain-membranes is comparatively frequent, but in the great majority of cases it is due to the presence of tubercles in the pia mater. In the child suffering from meningitis delirium is usually not so marked a symptom as is stupor or coma. This is owing partly to the impressible nature of the cerebral cells during early life, which leads them to be overwhelmed by an irritation which would in the adult produce only an active delirium, and partly to the tendency to the outpouring of serous exudation into the cerebrum being much greater in youth than in age.

For the purpose of diagnosis it is better to study first the commonest form of meningitis in childhood,—*i.e.*, tubercular menin-

gitis. Before doing so, the tubercular affection as it occurs in adults requires some consideration.

Except in very rare cases, *tubercular meningitis* in the adult is secondary, developed as the result of a tubercular infection produced by tubercular or caseous degeneration of the lung or other distant organ, or coming on during convalescence from typhoid fever or other acute systemic affection. It may develop suddenly with violent psychical disturbances, which may continue for a few hours or days and then be lost in coma. The first marked symptom may be furious maniacal outbreaks, happening only at night, the patient during the day being entirely rational and free from any pronounced symptoms of cerebral disease.

Occasionally the tubercular deposit is so localized that the earlier symptoms are those of a focal lesion. Thus, cases have been recorded in which an aphasia was the first evidence of the disease; and a local spasm, or even local paralysis, or a sudden Jacksonian epileptic attack, may usher in the disease. In typical cases the attack begins with a headache, which may be very severe, and may be accompanied by marked anxiety, depression of spirits, and often psychical symptoms resembling those of insanity, such as hallucinations, melancholia, or a mild mania. The motor symptoms usually follow rapidly upon the other evidences of cerebral disturbances. General or local convulsions are rare, but various forms of paralysis are frequent; the palsy may affect one arm or one leg, or take the form of a hemiplegia; under these circumstances it is rarely, if ever, complete. Ptosis, strabismus, dilatation or contraction of the pupil, facial palsy, or other losses of power about the face are very frequent, on account of the tendency of the tubercular exudation to mass itself about the base of the brain. Fever is usually a pronounced symptom, and may be irregular or may have a diurnal rhythm similar to that of typhoid fever. The abdomen is usually retracted, and constipation pronounced. Vomiting is often, but not invariably, present. If there be local disease of the alimentary canal, severe diarrhœa may entirely mask the other abdominal symptoms. Whenever in a case of phthisis, or during the convalescence from an acute constitutional disorder, symptoms of irregular cerebral disturbance develop, the physician should always suspect the occurrence of a tubercular meningitis. The symptoms of the disorder vary

greatly, and the diagnosis is justified whenever in the presence of the exciting cause organic brain-disease is indicated by the occurrence of headache, marked psychical disturbance, or local palsy, provided no other explanation of the symptoms can be made out.

The symptoms of a typical case of tubercular meningitis occurring in childhood may well be arranged in three stages besides the prodromic period. It must be borne in mind that this division is arbitrary, and that the stages insensibly pass into one another in any individual case; also that in some cases one or more of these stages are absent, and that in other instances the symptoms are so mixed together that none of the stages can be clearly made out. The first period is prodromic. The child's health begins to fail mysteriously; its disposition alters so that it becomes peevish and especially irritable; its sleep at night is broken, sometimes delirious, sometimes interrupted by night-terrors; the appetite fails, the bowels are constipated, and vomiting may occur: at the same time there is a feverishness rather than a distinct fever. This prodromic condition lasts for about a week, when the child enters the first stage of the developed affection. The symptoms of this stage are as follows: headache, which may not be severe, but which is commonly by older children bitterly complained of, or in young children is manifested by a peculiar plaintive cry, occurring at irregular intervals, and often breaking out in the midst of a restless night-slumber, although rarely heard after the coma has been fully developed, and so characteristic as to be known as the *hydrocephalic cry;* vomiting, which may occur only at long intervals, or may be incessant and associated with nausea, and which is very frequently produced by changes of position; constipation, with retraction of the belly,—a symptom which, however, may be entirely masked if there be tubercular or other irritation of the intestines or their glands; a condition of the pulse which is not in accord with the extent of the fever, and may be distinctly slow and even somewhat irregular; spasmodic contraction of the muscles of the neck, causing some retraction of the occiput and a perceptible stiffness of the neck when the head is raised from the pillows; fever, which has nothing characteristic about it, but is rarely severe. General or local convulsions may occur, and the pupil may be implicated. When, however, pupillary symptoms become marked, and the evidences of paralysis appear about the

eye and face, the child may be considered to have entered into the second stage of the disorder.

Stupor, coma, and muscular relaxation are the most pronounced symptoms of the second stage. The pupils are dilated or contracted, sluggish in their movements, or altogether fixed; strabismus, distortion of the face, failure of the power of articulation, or other evidences of loss of power in the muscles about the head, may show that one or more of the nerves at the base of the brain are suffering from the pressure of the exudation. Headache, vomiting, and constipation may continue. The pulse is usually slow and intermittent or otherwise irregular. Various contractures may be present; convulsions or convulsive movements of the extremities are frequent; the rigidity of the neck persists; and the hydrocephalic cry indicates that in the midst of the stupor the little patient is still sensible of his sufferings. As this stage progresses, the stupor deepens, until the child no longer exerts any control over the bladder or rectum and cannot be aroused. Gradually as the coma becomes more pronounced the final paralysis is reached, and often the rigidity of the neck and the retraction of the belly disappear amidst the universal muscular resolution. The pulse becomes rapid, feeble, and irregular; the temperature may become subnormal, or it may rise very high, or it may rise and fall without regularity or order until at last death ends all.

The symptoms of the prodromic stage of tubercular meningitis are sufficient to excite suspicion, but rarely do they warrant a positive diagnosis. If the case has developed in an ordinary manner, and especially if there is in the medical history of the child's family a distinct tubercular taint, the character of the disease is usually apparent by the time the first stage of the disorder is fairly entered into.

In irregular cases the diagnosis is not so easy. The prodromic stage may be slight, and there seems indeed to be a form of the disease in which the attack is said to be ushered in by convulsions. There is said to occur in young children an idiopathic meningitis or leptomeningitis, the diagnosis between which and tubercular meningitis may be attended with much difficulty. The idiopathic affection more frequently than the tubercular disorder begins abruptly with furious convulsions, but for several days

before this outbreak there may be headache, restlessness, sleeplessness, or delirium at night, change in the disposition, vomiting, constipation, contracted pupils, with excessive sensitiveness to light and sound, or other hyperæsthesia, vertigo, and other symptoms closely resembling those of the prodromic period of the tubercular disease.

During the convulsions of idiopathic meningitis the child is entirely unconscious: spasm of the glottis, as indicated by long-drawn, crowing inspiration and impeded expiration, is not rare. Either through it or through cramp-arrest of the respiration cyanosis may be induced and death occur during the first series of convulsions. Paroxysms of convulsions may succeed one another at short intervals for many hours, or may subside, when stupor or coma, ocular and facial paralysis, loss of sight and hearing, and progressively increasing muscular relaxation, with or without contractures, indicate the constantly-increasing pressure from exudation into the membranes and ventricles of the brain.

Since tubercular meningitis almost of necessity ends in death, whilst in non-tubercular meningitis the child has a chance of recovery, great interest attaches to the diagnosis between the two affections. Unfortunately, there are no symptoms which are pathognomonic of either disease. The existence of a known hereditary taint renders the diagnosis of tubercular meningitis probable; its absence favors the hope that the attack is not tubercular. A prolonged prodromic period indicates a tubercular affection; and yet I have seen three cases in which the symptoms seemed to warrant the diagnosis of a hopeless tubercular meningititis, but in which the patients recovered.

In one of these cases the father was a very dissipated man, and sufficient ground was afforded to warrant the suspicion that the meningitis was due to inherited syphilis. The second case occurred in an orphan fourteen years of age, of no known family history, but did not present any other symptoms of syphilis than those of a slow, progressive basilar meningitis. Recovery under the use of iodide of potassium was, however, complete.

In the third case, occurring in a child whose joints and ribs indicated a rachitic tendency, syphilitic taint was absolutely denied by both parents. There was, however, a history of possible traumatism, and the symptoms were chiefly furious repeated convul-

sions, with some rigidity of the base of the neck, rapid loss of flesh, and headache. Recovery took place under proper hygienic measures and the use of iodide, phosphates, etc., the iodide of potassium seeming to achieve most of the result.

Cerebro-spinal meningitis attacking a young child may produce symptoms which are not to be distinguished from those attributed to a fulminating idiopathic meningitis, and it is probable that cases supposed to have been idiopathic have really been instances of the epidemic disease. Moreover, pneumonia may produce symptoms closely resembling those of a true meningitis: even in the adult the cerebral symptoms of a pneumonia may completely mask the pulmonic disturbance, and Grisolle, as quoted by Prof. Loomis, affirms that the usual physical signs may be altogether wanting. I have seen a number of cases in which the sole distinct symptomatic evidence of pneumonia was acceleration of the breathing, noticeable only when carefully looked for, and in which no crepitant râle could be heard at any time during the disease. Absence of vesicular murmur or presence of bronchial breathing, however, usually betrays the pulmonic lesion. Possibly bronchial breathing may in some cases be wanting, and even a transmitted vesicular murmur be heard; but I can scarcely conceive that percussion-dulness can be absent in the pneumonic consolidation of the adult. In young children I have seen headache, strabismus, convulsions, intense persistent rigidity of the neck, with fever and a slight occasional barking cough, followed by death on the sixth day, and at the post-mortem have found extensive pneumonia, with simple hyperæmia of the brain-membranes and an excess of serous fluid in the brain, the microscope showing that there had been no out-wandering of blood-corpuscles and no purulent or fibrinous exudation. In this case the only physical sign that could be detected was a relative dulness over the affected lung. The percussion-note was distinct and clear, but not quite so clear as over the opposite lung. In such a case as this the lung-affection might very easily be overlooked. The intensity of the fever is, however, much greater than in either meningitis or cerebro-spinal meningitis, and is especially out of proportion to the severity of the meningeal symptoms; the breathing is also excessively accelerated. Meningitis, generally of the vault, is a not very infrequent complication of pneumonia, but there is no way of

distinguishing between meningitis and meningeal irritation in the pneumonia of childhood. The practical points are that in these cases the pneumonia is the primary affection, to which the treatment is to be especially directed, and that, whenever symptoms of fulminating meningitis appear in children or very old people, the lungs should be carefully examined, especially in their apical lobes, which are usually, but not always, the part affected in the so-called cerebral pneumonia.

Acute Perioncephalitis,—Acute Peripheral Encephalitis, Phrenitis, Mania Gravis, Typhomania, Acute Delirium, Delirium Grave, Bell's Disease (Luther Bell).—This affection sometimes follows intense emotional excitement, sometimes appears as the result of a prolonged strain upon the nervous system, such as occurs during a business crisis, and sometimes develops without apparent cause. It is more frequent in females than in males, and especially occurs in cases of seduced pregnant women.

The symptoms may come on with extreme suddenness, or may be preceded by prodromic evidences of cerebral disturbance. These prodromes in rare cases take the form of increase of mental power, in others of brief nocturnal attacks of wandering delirious restlessness; or there may be short periods of impaired consciousness, especially upon waking in the morning, or, as in one of my cases, even an epileptiform convulsion. The fully-developed disorder naturally divides itself into two stages,—first, that of acute maniacal delirium, and, second, that of apathy and collapse, with coma. The delirium is always of an excited type, accompanied by violent incoherent speech, and usually by a fury of fighting and of destructiveness.

Hallucinations and half-formed delusions are present, and often bear a close relation to the cause of the attack. The abandoned mistress will in her ravings recount her past shame and present agony. The business-man will be perpetually occupied with an incoherent jumble of business transactions. Almost invariably along with the delirium there is great physical restlessness, which grows more intense until it causes the patient to leap from his bed and to attempt to run away. Very commonly violent assaults are made upon the attendants. Convulsions are not common. In many cases the delirium is at first not continuous, occurring only at night, or is at least interrupted by brief intervals of comparative

rationality during the daytime. Finally, however, there is persistent intense mania. In one of my own cases the patient, during the day, told his wife that she must protect herself from him,—that he loved her most fondly, but that he was going into a condition of insanity, in which he would certainly kill her. From this time until his death he was furiously maniacal during the night, although for several days he would recognize his friends during the daytime, and for a moment or two talk rationally. There is usually absolute insomnia. The pulse is rapid, and even if in the beginning it possesses a show of force, it is really soft and compressible. There is no desire for food, and generally an absolute refusal to take it. There is also distinct fever, the temperature rising sometimes to 106° F. According to my observation, the temperature varies with a stormy irregularity which is almost characteristic, rising and falling many degrees many times during the twenty-four hours. Its variations are connected with the mental and physical excitement of the patient,—maniacal outbursts producing an immediate rise of the temperature. The pupils may be contracted, dilated, or normal. In the course of a few hours to several days the second stage of the disorder develops. There is now quiet, with coma or else muttering delirious unconsciousness, failing pulse, cool skin, and general evidences of collapse. In the early part of this stage, when aroused the patient may respond incoherently, or perhaps give some slight evidences of comprehending what is said to him, but rapidly sinks lower and lower until he dies from exhaustion. Early in the disorder the skin becomes very harsh, and finally cyanotic; in the later stages irregular desquamation, or even ulceration, may occur. In a case quoted by Spitzka the anæsthesia was so complete that the patient gnawed off a portion of one of his fingers. Pemphigus-like vesicles, phlegmons, gangrenous patches of skin, or gangrenous extremities, not rarely appear, but are frequently absent, and are not characteristic. Complete recovery never takes place, although it is affirmed that in rare cases the patient is restored to a fair degree of physical health with only a slight mental defect. Usually the end is death; sometimes permanent complete dementia and more or less wide-spread paralysis result. After death evidences of peripheral encephalitis are to be found.

This disease is very closely related to acute mania: indeed, unless it be by the presence of marked fever, and by the intensity of the symptoms, I do not see how the affection is to be separated from acute mania. If it be correct, as is asserted by Hammond (*Treatise on Insanity*, p. 546), that the temperature is never elevated in acute mania, then it is possible to diagnose between acute mania and peripheral encephalitis. Folsom, however, affirms that in acute mania the skin is hot; and at present we are not able to state positively that attacks of acute mania are other than cases of peripheral encephalitis of a mild type. (See Acute Mania, p. 471.)

General Paralysis of the Insane—Paretic Dementia, Periencephalitis, General Paralysis of the Insane, Paresis—is a disease in which the lesion is a progressive inflammatory alteration of the brain-cortex, which registers itself symptomatically in the motor, sensory, and mental spheres of action.

Cases of paretic dementia are divisible, so far as their mental symptoms are concerned, into four varieties; but it must be remembered that this division is an arbitrary one, and that whilst abundant cases exist in nature corresponding accurately to one or other of the classes, yet every grade of case exists between the classes, and the march of the mental malady sometimes is so irregular that in one portion of its course the individual case might be assigned correctly to one variety of the disease, but at another time would belong to another variety.

In the first form of paretic dementia are included those cases in which progressive failure of power constitutes almost the whole mental disturbance, the mental faculties consentaneously growing less and less until the patient becomes childish, and at last completely demented, without emotional disturbance or delusions having been present. (It is these cases especially that are popularly spoken of as softening of the brain.)

The second variety of paretic dementia is that in which delusions of grandeur or expansive delirium are present. The character of these delusions has already been sufficiently pointed out. (See page 431.) It is essential to remember that these delusions may exist in so mild a degree that they may be very readily overlooked. Further, in many cases they are replaced by a *bien-être* which may be looked upon as a condition of undeveloped delu-

sion. Thus, the man sunk in the deepest poverty will be excessively happy and jolly, misfortunes having no power to depress him, although he makes no assertion of the possession of great power or wealth. In all cases of the present variety of general paralysis there is progressive mental failure, and it is, therefore, evident that the cases in which a simple *bien-être* exists may be looked upon as midway between the first and the second variety of the disease.

Maniacal outbursts may occur in any variety of general paralysis, but they are more common and more frequent when there are delusions of grandeur.

The third form of general paralysis is that in which there is emotional depression, and even pronounced melancholia, with depressive delusions. Not rarely the depressive delusion relates to the person of the patient, who believes himself ill, deformed, or wanting in some member or function. In this way arises the so-called hypochondriacal variety of general paralysis.

The fourth form of general paralysis is that described by Dr. Fabre, in which excitement and depression alternate so as to make a periodic or circular insanity. The existence of this variety has been confirmed by Dr. W. Julius Mickle (*General Paralysis*, London, 1880), who further says that when there are only two phases these succeed each other suddenly, but that in some cases there are three periods,—(1) excitement, (2) calm, (3) depression, —in this differing, therefore, from non-paralytic circular insanity, in which the usual order is (1) excitement, (2) depression, (3) quietude or lucidity.

The physical symptoms of paretic dementia are chiefly connected with the motor function, although late in the disorder sensation is also impaired, and may be almost abolished. Except in regard to the epileptic attacks (see page 116), the motor symptoms are always paralytic, and are especially characterized by their incompleteness and by their connection with tremors and loss of control over muscular movements. In the earliest stages of the disorder the loss of control over complicated muscular movements is first manifested in the hands, and may be very pronounced at a time when the general muscular power is but little weakened. Thus, a man may be able to lift many pounds, although he cannot write his own name. The acute development of such a loss

of muscular control occurring in a man of middle age, without obvious cause, is a serious symptom, and probably, in the majority of cases, is prodromic of general paralysis. It is especially to be noticed very early in engravers and other persons whose daily vocation requires great technical skill.

A varying inequality of the pupils may occur very early, although more constant in the later stages of the disease. It may be associated with excessive dilatation or contraction. When there is no focal brain-lesion, and no disease of the neck or of the cervical spinal cord, this symptom is almost pathognomonic.

The departure of the speech from the norm in general paralysis is partially of mental and partially of physical origin. As a consequence of the loss by the lips and tongue of their delicacy of movement, there is a difficulty of pronunciation, which is especially manifested with lingual and labial consonants and in the syllables of long words. This causes a peculiar stuttering or hesitation, with some thickness of speech and an occasional elision of syllables, so that the speech somewhat resembles that of intoxication. In advanced stages of the disease the uncertainty of the movements of the lips and tongue is plainly visible to the eye, and is associated with tremor, or, more correctly, with tremulousness. In general paralysis the mind thinks slowly and imperfectly: it fails not only in the formation of ideas, but also in the quick association of these ideas with suitable words. There is, consequently, slowness as well as hesitation of speech. In some cases the mental actions seem to be performed in a rhythmical manner, giving rise to a peculiar utterance which somewhat resembles that used by the school-boy in scanning Latin poetry, and hence often spoken of as the "scanning speech." There is also in many cases a use of improper words. Not rarely the paralytic talker drops a word from his sentence or repeats a word; mayhap he elides or repeats a whole clause. Movement of the jaws similar to mastication may take place, and even cause grinding of the teeth or champing of the jaws.

The loss of adroitness and exactitude of movement may first appear in the hands. The handwriting becomes shaky and irregular, and the letters are ill formed, even widely separated from one another, sometimes resembling hieroglyphs rather than members of the Roman alphabet. Very frequently the finely-graded

strokes of correct writing disappear in a common, thick, uncertain line. The writing not only shows the physical degradation, but has the same mental characteristics as the speech. The ideas are often incongruous and devoid of proper association, and the words incorrectly used. Letters are dropped out, syllables omitted or repeated, and words or even clauses elided or interjected.

The gait may be early affected. It becomes awkward and uncertain, the steps may be long and slightly irregular, and the patient's lack of control over his movements comes out sharply when he attempts suddenly to turn or to alter his position. As the disease progresses, the gait becomes slow, heavy, and unsteady, whilst the widely-separated feet readily trip over an inequality or unexpected obstacle. In the advanced stages the posture of the patient resembles that of old age, the body being bent awkwardly forward or to one side. With difficulty he walks with a slow, unsafe, swerving gait; in the most advanced stages tottering forward, aided by an arm or some support, and day by day losing control over his limbs, until he becomes bedridden.

The symptoms of general paresis may be summed up to be—change of character; progressive mental deterioration, with delusions of grandeur, emotional exaltation, or emotional depression; occasional maniacal outbreaks and epileptic attacks; progressive physical deterioration, as shown by irregularity of the pupils, disorder of speech, loss of control over the movements of the hands and legs: all symptoms finally being swallowed up in a complete paralysis of intellection and of voluntary motion. When the disease is fully formed there can be no difficulty in recognizing it. In the earlier stages, however, the diagnosis may be almost as difficult as it is important. In making it, the age of the patient and the presence in the history of the ordinary causes of general paralysis should have great weight.

In civil life the affection is most frequent between forty and fifty years of age, although it not rarely occurs as early as thirty years, and more frequently as late as the fifty-fifth year. In sailors and soldiers Mickle noted that the average age was about thirty-three. The causes of the affection appear to be habitual emotional and, to a less extent, purely intellectual over-excitement; excessive use of alcohol; sexual excesses, especially when combined with syphilis; and, much more rarely, sunstroke and

other traumatisms. Whenever any of the earlier symptoms appear in a man of middle age whose life-history presents the causes of the disease, general paralysis should be considered imminent, and, whilst it may not be proper to give a positive opinion, it is usually right to take measures of restraint. The earliest important symptoms are an apparently causeless change of character, epileptic attacks which cannot be otherwise explained, and distinct mental symptoms such as have been described. When the diagnosis rests between general paresis and some other form of mild insanity, the condition of the memory should be carefully studied. Under these circumstances distinct failure of memory points very strongly towards general paresis. Among the physical symptoms the most important are inequality of the pupils, and loss of power of executing fine movements, such as those of writing, of buttoning or unbuttoning the clothes, of dancing, etc. These physical symptoms usually come on early; some of them may even precede disturbance of intellection. On the other hand, I have seen the physical health greatly improve and the muscular power increase in the earlier stages of the disorder,—at a time, too, when the intellectual symptoms were very pronounced.

Cerebral Sclerosis.—Sclerotic affections of the brain more usually take the form of a multiple sclerosis than that of a widespread cortical change. In multiple selerosis the only mental aberration that is common is a progressive loss of power, especially marked, at least in the earlier stages of the disease, in regard to the memory of recent events. If considerable cortical regions be involved, complete dementia may result even in a disseminated selerosis. In terminal dementia it is not uncommon for sclerotic changes to be detected at the autopsy, and it is probable that various cases supposed to be instances of pure insanities are really examples of the earlier stages of a cerebral selerosis. At present, however, we are not able to connect mental aberration with incipient selerotic disease of the brain-cortex, or in any way to diagnose the existence of the latter, unless it manifests itself also by physical symptoms. For further remarks upon this subject, see Syphilitic Insanity, p. 464.

DIATHETIC INSANITIES.

The diathetic insanities are not distinct forms of disease, but groups of symptoms of various and varying character, which are the outcome of constitutional vice or disease. Thus, there is nothing in the symptoms of a gouty insanity which would enable us to diagnose the nature of the case. The cause of the mental aberration in such a case can be recognized only by recognizing the presence of lithæmia. The importance of distinguishing an insanity of the present class lies in the fact that relief is to be obtained not by treating the insanity, but by treating the diseased condition which is the cause of the mental disorder.

The most important of the diathetic insanities are the Gouty, the Epileptic, the Hysterical, and the Toxæmic.

Gouty Insanity.—It is well known that gouty paroxysms are frequently accompanied and preceded by peculiar nervous irritability. At such times there is a depression of spirits, with an irritability so great that it can scarcely be controlled by the patient. In some cases these symptoms become so intensified as almost to amount to insanity; moreover, hallucinations, delusions, loss of mental power,—indeed, almost every conceivable manifestation of mental disorder,—may be directly or indirectly caused by gout. Carrol in 1859 said, "Gouty mania is occasionally seen;" and in 1875, Dr. P. Berthier (*Des Névroses diathésiques*, Paris) published a collection of forty-six cases of nervous disease attributable to gout; one of hallucinations; one of migraine; four of tetanus; three of chorea; one of hypochondria; seven of epilepsy; one of paralysis; and twenty-six of mental affections, including in these dementia, melancholia with stupor, mania. Although in some of these cases the evidence is not at all positive that gout was the *materies morbi*, yet in others the relation seems to have been clearly made out.

In his paper read before the International Congress at London, 1881 (iii. 640), Dr. Raynor supported the following conclusions:

1. Protracted gouty toxæmia, when not very intense, usually results in sensory hallucinations or melancholia.

2. Sudden and intense toxæmia results in mania or epilepsy.

3. Intense and protracted toxæmia usually results in general paralysis.

4. If there be a tendeney to vaseular degeneration from plumbism, alcoholism, etc., varying degrees of dementia are produced.

In the diseussion whieh followed the reading of Dr. Raynor's paper, Drs. Savage and Criehton Browne, of London, both expressed the belief that gout does cause insanity, the latter, however, qualifying by the statement, "only where there is hereditary predisposition to insanity."*

The conclusions of Dr. Raynor are borne out by a ease of my own. A lady at regular intervals of four years had had a number of attacks of severe gout associated with great depression of spirits at times amounting almost to pronounced melancholia. Finally, at the end of four years of health, the patient was seized with symptoms of aeute dementia or stuporous melaneholia, associated with marked tenderness of the nerve-trunks, and, in eertain portions of the body, violent neuralgia, and a urine that was loaded with uric acid and urates. Death oeurred after some weeks from œdema of the lungs. At the autopsy there was found gouty kidney and a remarkably pronounced atheromatous degeneration of the eerebral vessels, the lumina of some of the basilar arteries of the brain being almost obliterated.

Epileptic Insanity.—In eonsidering the relations of epilepsy to mental aberration it is necessary to diseuss separately the mental symptoms whieh may oeur in or replaee a single paroxysm, and those whieh are the result of a long suecession of paroxysms. The paroxysmal mental symptoms may be eonsidered under the heading of Epileptie Automatism and Epileptie Mania. In a very large proportion of cases the epileptie paroxysm is followed by profound sleep, from which the patient wakes in a more or less dazed condition, which may continue for some moments. In many eases the patient may, after the epileptie paroxysm, appear to be conseious and yet not really be himself, as is shown by his subsequently being unable to remember anything that has happened shortly after the eonvulsion. In some cases distinct hysterical manifestations aceompany this post-epileptic eondition, and even hysterical eonvulsions may occur.

* Further proof of the connection between gout and insanity may be found in the Paris Thesis of M. Belliard (1882, No. 269), in which are detailed various cases.

The so-called automatic actions of epilepsy are probably in the majority of cases post-paroxysmal, and occur during the period which has just been spoken of. In their simplest form these actions consist in the doing of something which is usually incongruous. According to Gowers, a very common performance is that of undressing. In other cases the patient habitually lays hold of all small objects near him and secretes them about his person. In a case related by Gowers, the patient was accustomed to cut bread, butter it, and eat it as fast as possible. Sometimes the actions are very complex. Gowers relates the case of a carman who, for an hour after his fit, would drive through the most crowded streets of London without accident.

Sometimes the subject of epileptic automatism will get up after the convulsion and continue with whatever work was in hand when the attack developed, although, in fact, perfectly unconscious. Thus, a woman under my own care has been attacked with the convulsion as she was setting the table for a meal: getting up in two or three minutes, she would continue to dish up the dinner, arrange the plates, etc., in an apparently natural way, but would after a time suddenly wake up and have no knowledge of what she had done. Other cases of epileptic automatism have already been cited (see page 107), and sufficient examples given to show that the acts may apparently be purposive or purposeless, and most simple or most complex, and that often it is very difficult to persuade by-standers that the patient is not in a condition of true consciousness. In many cases of epileptic automatism no display of emotion is made; sometimes, however, the patient is hilarious, and even aggressively affectionate, and still more frequently rage or violent emotions are manifested. It is through cases in which violent passion asserts itself that epileptic automatism passes into the so-called epileptic mania,—which, indeed, may be very logically considered as a form of the automatism associated with excited emotions.

Epileptic automatism is more apt to follow paroxysms of petit mal than the major convulsions. Not rarely the vertiginous and convulsive symptoms are so slight that they are discoverable only by the most careful observation; and it is probable that in some cases the automatic actions are the sole outcome of the epileptic discharge, all the other stages of the paroxysm being wanting. I

have certainly seen the attack of epileptic automatism precede the general convulsion.

The actions of epileptic automatism, as already stated, may so closely resemble those of the normal state as to make the recognition of their true nature somewhat difficult. The characteristic feature is, however, that the patient does not after his recovery remember anything of the occurrences which have taken place during the automatic state. The period is an absolute blank with him. It is hardly necessary to point out the close relations which exist between the epileptic automatism and the so-called double consciousness. The fact that in typical double consciousness the subject shows vivid recollection of occurrences during previous paroxysms of the same type affords some distinction; but in nature cases appear to grade into one another, the connecting remembrances being sometimes very slight and sometimes very vivid.

In *epileptic mania,* so called, there is violent excitement and delirium, which may take the form of an acute mania or of an agitated melancholy: in either case the incoherence is usually less than in the corresponding non-epileptic affection. Not rarely after a primary period of violent disconnected speech the patient is seized with an ambitious or mystic delirium, or sometimes a delirium of persecution, or, more rarely, with an erotomania, in which sentence after sentence flows out with extraordinary volubility. The attack usually comes on suddenly, and is always accompanied by hallucinations, which sometimes develop brusquely, or, more rarely, in the course of a few minutes. The hallucinations affect all the senses and give rise to delusions which conform with the type of the emotional disturbance. The delirium may last for a few moments to several days. It is especially characterized by the tendency to acts of extreme violence,—to suicide in the melancholic form and to homicide in the maniacal variety.

In epileptic fury the subject has no control over his actions, and when murder and other crimes are committed it is important that the medical jurist recognize the true nature of the attack. When the mania is of mild type the danger of overlooking its character is greatest. The diagnosis is to be made by obtaining the history of previous attacks of epilepsy, by the brutality and causelessness of the crime, and especially by the fact that the

patient has no memory of occurrences which took place during the mania. In a certain proportion of the cases the attacks of epileptic mania are repeated in exact counterfeit one of the other. The maniacal outbreak, may, however, not recur for a great length of time. The difficulties of the expert are increased by the fact that the first paroxysm of an epilepsy may take the form of a furious outbreak of epileptic mania. Under these circumstances it may be essential that the patient be kept for a length of time under surveillance, since, although the circumstances of the paroxysm may satisfy the mind of the medical expert, they may fail to carry conviction to judge and jury. Esquirol states that the homicidal mania of epilepsy is never radically cured, and that its subject is always liable to a fresh outbreak. Whether this be absolutely true or not, it is certain that the recurrence is sufficiently habitual to demand the perpetual surveillance of the epileptic criminal.*

Epilepsy frequently leads to mental degradation, which may end in complete dementia. More rarely a permanent insanity develops in the epileptic, although it is doubtful whether the convulsions in these cases are not the direct outcome of the original neurotic vice, rather than the cause of the insanity. The type of such insanity is said to be usually melancholic, with delusions of persecution and suicidal impulses. The characteristic mental state of chronic epilepsy is progressively lowered mental power, with a peculiar irritability and brutal selfishness, and outbreaks of furious anger on the slightest provocation. Even while the mental powers are still active, epileptics very frequently are peculiarly irritable and revengeful. After a paroxysm these tendencies are increased. The tendency of epilepsy to cause dementia is usually in direct relation to the earliness of the age at which it first appears, as is shown in the following analysis of fourteen hundred and fifty cases collected by Gowers:

Age of First Appearance.	Mental Defect developed.
Under 10 years	55 per cent.
Between 10 and 19 years	36 " "
" 20 and 30 years	3 " "
Over 30 years	6 " "

* For further details upon this subject consult M. V. Magnan, *L'Épilepsie*, Paris, 1882.

Hysterical Insanity.—Severe hysteria is usually accompanied by a peculiar mental organization, which may amount to a distinct and characteristic psychosis. In its aggravated form this psychosis ought to be considered as belonging to the partial insanities, and ought to acquit its victim of legal responsibility and to afford sufficient grounds for restraint. The peculiar characteristics of this hysterical temperament have been so vividly set forth in a few sentences by Dr. Folsom that we quote his words:

"It is characterized by extreme and rapid mobility of the mental symptoms,—amnesia, exhilaration, melancholic depression, theatrical display, suspicion, distrust, prejudice, a curious combination of truth and more or less unconscious deception, with periods of mental clearness and sound judgment which are often of greater degree than is common in their families; sleeplessness, distressing and grotesque hallucinations of sight, distortion and perversion of facts rather than definite delusions, visions, hyperæsthesias, anæsthesias, paræsthesias, exceeding sensitiveness to light, touch, and sound, morbid attachments, fanciful beliefs, an unhealthy imagination, abortive or sensational suicidal manœuvres, occasional outbursts of violence, a curious combination of unspeakable wretchedness alternating with joy, generosity and selfishness,—of gifts and graces on the one hand and exactions on the other. The mental instability is like a vane veered by every zephyr. The most trifling causes start a mental whirlwind. There is no disease giving rise to more genuine suffering or appealing more strongly for sympathy. Yet when this is freely given it does harm. One such person in the house wears out and outlives one after another every healthy member of the family who is unwisely allowed to devote herself with conscientious zeal to the invalid."

During the paroxysm of major hysteria there is a period of delirium which may simulate acute mania, and I have seen recurring attacks of hysterical epilepsy replaced by a furious outbreak of acute mania, lacking in none of the symptoms characteristic of that disease. It seems to me that in such a case the maniacal explosion must be looked upon as the direct outcome of the hysterical neurosis, and that therefore the existence of an hysterical acute mania not in itself distinguishable from ordinary acute mania must be acknowledged. In most cases in which such maniacal symptoms exist the neurosis is so thoroughly engrafted

upon the constitution that permanent recovery is not possible, the patient during life suffering from various forms of hysterical attack, and being always possessed of the peculiarities which have already been spoken of as characteristic of the hysterical temperament. Hysterical symptoms may occur during almost any form of insanity, but do not warrant our looking upon such a melancholia or mania, or whatever form the affection may take, as hysterical, scarcely more than we should be warranted in considering pneumonia when associated with hysterical symptoms as hysterical. At the same time, the relation of the hysterical temperament to monomanias and to general insanities is distinct, and, according to my belief, it is entirely possible for any form of insanity to be simulated by symptoms which have their origin in the original faulty organization that is the basis of chronic hysteria: moreover, such faulty nerve-organization is closely allied to the peculiar neurotic temperament which is the basis of much insanity.

Syphilitic Insanity.—Insanity of any type may occur without definite organic brain-disease in a person who has syphilis. In such a case the syphilis, by causing mental distress or general failure of health, may be a potent factor in the production of the mental disease; but there is at present no reason for believing that syphilis can directly produce insanity without a demonstrable brain-lesion. By interfering with the circulation of the brain-cortex, or by propagation of the inflammation to the cortex, gummatous meningitis may profoundly influence the brain-functions, and experience has shown that the aberrations produced by these organic changes may simulate almost any form of insanity. Such insanity offers no characteristic symptoms, and really belongs to the complicating insanities. The significance of the mental disturbance is to be made out by recognizing the physical symptoms of the organic lesion. Violent headache, epileptic attacks, ocular or other forms of local palsies, local spasms, localized neuralgic pains, or other evidences of generalized or localized gummatous inflammation, almost invariably enable us to make out at once the nature of the disease. Syphilis may, however, produce a wide-spread structural disease of the brain-cortex without implication of the membranes or of the basal nerves, and the connection between the consequent mental derangement and the syphilitic infection

may be very difficult to trace. There may be no symptoms of a focal disease of the brain, and, indeed, no distinct proof of the existence of gross organic lesion.

Sometimes the insanity is maniacal; now it takes the form of religious melancholy, again it resembles confusional mania, rapidly passing into dementia. There is certainly a form of sclerosis of the cerebral convolutions which has a more or less direct connection with syphilis, in which the symptoms during life are those of a chronic insanity with a gradual deterioration of the mental powers, ending in complete dementia. In one case in which I had the opportunity to follow the symptoms for a long time during life, and to confirm the diagnosis by a post-mortem examination, there was no headache, but for many months a peculiar mental condition marked by great restlessness, with a perpetual desire to be upon the go, with excessive volubility and a curious loss of the power of judging of the relative importance of things, so that the man would talk for hours about a trifling incident and have no interest in events of the utmost importance. In the course of time wild maniacal symptoms were added, and the case passed into an apparently ordinary dementia.

Although medical records prove that a patient whose symptoms are apparently those of a pure insanity may have a syphilitic brain-disease which will yield to treatment, such cases are extraordinarily rare. In an experience covering several hundred cases of brain-syphilis I have never seen one. I have seen a number of attacks of an apparent pure insanity in persons who have had syphilis, but have never been fortunate enough to get good from antisyphilitic remedies. It is otherwise with cases whose symptoms resemble those of general paralysis of the insane. I think we must recognize as established the opinion of Voisin,* that there is a syphilitic periencephalitis which presents symptoms closely resembling those of general paralysis. Such cases are examples of the *pseudo-paralysie générale* of Fournier.† The question as to the diagnosis of these cases from the true incurable paresis is very important, and has been considered at great length by Voisin,‡ Fournier,§ and Mickle.‖ The points which have

* *Paralysie générale des Aliénés*, 1879. † *La Syphilis du Cerveau*, Paris, 1879.
‡ Loc. cit. § Loc. cit. ‖ *Brit. and For. Med.-Chir. Rev.*, 1877.

been relied upon as diagnostic of syphilitic pseudo-general paralysis are—the occurrence of headache, worse at night and present among the prodromes; an early persistent insomnia or somnolence; early epileptiform attacks; the exaltation being less marked, less persistent, and perhaps less associated with general maniacal restlessness and excitement; the articulation being paralytic rather than paretic; the absence of tremulousness, especially of the upper lip (Fournier); and the effect of antispecific remedies.

When the conditions in any case correspond with the characters just paragraphed, or when any of the distinguishing characteristics of brain-syphilis, as previously given, are present, the probability is that the disorder is specific and remediable. But the absence of these marks of specific disease is not proof that the patient is not suffering from syphilis. Headache may be absent in cerebral syphilis, as also may insomnia and somnolence. Epileptiform attacks are not always present in the pseudo-paralysis, and may be present in the genuine affection; megalomania may be very pronounced in specific insanity. A case with very pronounced delirium of grandeur, in which the autopsy revealed unquestionably specific brain-lesions, may be found in Chauvet's *Thesis*, p. 31. I have seen symptoms of general paralysis occurring in persons with a specific history in which of these so-called diagnostic differences the therapeutic test was the only one that revealed the true nature of the disorder. In these cases a primary, immediate diagnosis was simply impossible.

In conclusion, I may state that it must be considered as at present proved that syphilis may produce a disorder whose symptoms and lesions do not differ from those of general paralysis; that true general paralysis is very frequent in the syphilitic; that the only constant difference between the two diseases is as to curability; that the curable sclerosis may change into or be followed by the incurable form of the disease. As a careful antisyphilitic treatment can do no harm, in any doubtful case of insanity it should be essayed.

Alcoholic Mental Disorders.—Mental disturbances produced by abuse of alcohol may be divided into the subacute and chronic forms, to which the names Delirium Tremens and Alcoholic Insanity may be assigned.

Delirium Tremens.—Delirium tremens is a peculiar series of

acute symptoms which are produced by excessive drinking. The affection is especially apt to develop upon the sudden cessation in the use of the stimulants, but may come on during the debauch. In their mildest form the symptoms constitute that condition known by old drunkards as "the horrors," in which the sleep is disturbed, the hand tremulous, the mind weak and confused, and the patient troubled with frightful imaginings, vague alarms, and an apparently causeless depression of spirits. When the attack is more severe, hallucinations of sight, of hearing, and, more rarely, of touch, occur. These hallucinations always have in them an element of terror or of horror. Disgusting objects, such as snakes, toads, rats, and mice, and similar unclean creatures, crawl over the bed or the person. Voices predicting evil, or bringing messages of remorse, or uttering threats of punishment, are heard. The patient may seem violent, and may even attack his attendants, but the violence is that of terror, and not of aggression. The attack is an attempt at defence. There is great insomnia, and usually when the patient can be made to sleep the mind is clear after the awakening. This is not, however, invariably the case: I have seen delirium tremens gradually pass through successive days of wakefulness and nights of sleeping into a chronic mania not readily to be distinguished from that arising from other causes. In the earlier attacks of delirium tremens occurring in very robust people, when all the mucous membranes are irritated, and when probably there is direct irritation of the brain and its meninges, there may be a slight febrile reaction and even a strong and excited pulse; but the disease is typically asthenic, with loss of muscular power, tremulousness, and rapid feeble pulse, and when death occurs it is from exhaustion. Cardiac failure is in such cases always to be guarded against.

Sometimes the patient suffering from delirium tremens has sufficient rationality to receive his physician with a quiet, gentle courtesy, and to answer questions without irritation. It will be noted, however, that he is evidently preoccupied, and that occasionally he turns his head or casts furtive glances from one part of the apartment to the other; and a little finesse will reveal the fact that during the whole time he is seeing visions or hearing sounds, or is at least laboring under a profound apprehension of attack.

The diagnosis of delirium tremens is usually easy, even when the history of the case is not clear. The peculiar terror underlying all the delusions, hallucinations, and attempts at violence is characteristic, as is also the tremulousness of the hands when extended. When pneumonia occurs during a period of delirium tremens the type of the delirium may change, tremors may be lost, and the patient may become so violently aggressive as to lead to a mistaken diagnosis.

Alcoholic Insanity.—The prolonged use of alcohol may lead to a gradual functional and finally structural alteration of the nervous system. Under the continuous influence of the narcotic the brain performs its functions slowly and imperfectly and the mental movements become sluggish and weak; the memory is greatly impaired; the power of fixing the attention steadily diminishes, but the intellectual weakness is especially shown by the lessening of the power of the will, so that not only is the judgment uncertain but its dictates are not carried out. There is also a distinct tendency to emotional depression, and often a peculiar suspiciousness, which is the ground-work for delusions. A step further, and hallucinations haunt the victim. The route to insanity and irresponsibility from this condition is short. Out of such a state is easily developed the most characteristic and frequent form of alcoholic insanity,—namely, that with depressive delusions. In some cases this variety of alcoholic insanity appears suddenly with symptoms for a time not to be distinguished from delirium tremens. Indeed, I think it perfectly correct to say that a patient may pass from delirium tremens into alcoholic insanity.

It is affirmed that headache and other symptoms of sudden congestion of the brain occasionally usher in the attack of alcoholic insanity. When the symptoms are active, hallucinations* are very numerous, constantly changing, and almost always are such as to inspire terror or disgust. In a very short time they are accompanied by delusions of persecution: voices of reproach, threatening, or remorse, mocking faces, unclean beasts, tormenting devils,

* Spitzka says they are usually of vision; Dr. F. Lentz (*De l'Alcoholisme*) says that they are almost exclusively of hearing; my experience is that both forms of hallucination are frequent.

—these and similar visions drive the victims into profound melancholy, and finally may lead to suicide or murder. According to Spitzka, the delusions of chronic alcoholism almost always relate to the sexual organs, to the sexual relations, or to poisoning. Underlying this variety of alcoholic mania is frequently an intense fear, which may lead to violence, as when a man kills his wife because he fears that she will poison him. Not uncommonly the depressive sexual delusion leads to an outburst of uncontrollable jealousy and rage, so that wife-murder from motives of jealousy is not a rare result of alcoholic mania. There is in some of these cases a very marked relation between the presence of alcohol in the blood and the insane outburst. The drunkard may be, when not under the influence of the poison, fairly rational, but is converted by alcohol into a wild beast, although he has few or none of the ordinary symptoms of intoxication. The man may walk straight, and talk rationally on general subjects, but be profoundly under the influence of a depressive or persecutive delusion which disappears when the blood is free from alcohol. Thus, in the case of a man recently tried at Elkton, Maryland, for the murder of his brother-in-law and child, it was proved that when the prisoner had abstained for two or three weeks from liquor he was kind to his family, and attached to his wife, in whom he also had complete confidence: so soon as he began to drink again he would become possessed with the idea that "she was no better than a common whore," and on several occasions he had attempted to murder her. The immediate recovery of the man during abstinence removed his case from being fairly considered as one of insanity; but, although a few moments before the murder the man had been talking rationally, the court came to the conclusion that he was in a state of temporary insanity from the influence of alcohol,—that is, in the condition of legal drunkenness,—and that the degree of his crime was therefore manslaughter: it being the theory of the law that a sudden murder committed during intoxication is not committed with that malice prepense which is necessary to constitute a murder in the first degree. The prisoner had been drinking heavily for twenty-four hours: he first shot his brother-in-law in front of his house, then went in and called his two little children to himself, and blew out the brains of his oldest son whilst the child was on his knees praying for mercy.

The second son, seizing hold of his father's pantaloons, pleaded for his life, but, seeing that his father continued to load his gun, ran for the front door; as he suddenly stopped to open it, the father fired, the shot tearing away the brim of the boy's hat.

The relation between depressive alcoholic insanity and mania a potu is, as has been already stated, very close. Insomnia, emotional excitement, especially connected with fear, hallucinations, and delusions, are common to each; but the tremors are more marked in delirium tremens, and when an attack of alcoholic insanity is acute and tremors are pronounced, it may be considered to be mania a potu.

Dr. F. Lentz (*loc. cit.*, page 491) calls attention to a form of alcoholic insanity with expansive delusions and hallucinations of sight and hearing which, very strangely, in most instances relate to God and a future state. Visions of supernatural beings, and especially of the Deity bathed in an aureole of light, perpetually haunt the patient; the ministrations of angels seem to bring relief, or mayhap the voice of God himself is heard in command or instruction.

It would appear that two forms of alcoholic insanity must be recognized,—one a lypemania, or melancholia with delusions of persecution; the other a megalomania, with a strong tendency to religious hallucinations.

PURE INSANITIES.

In the present group are included those insanities in which there are no other evidences of brain-disease. The group includes, in other words, all cases of ordinary insanity, so called, in which there are no physical symptoms, and in which we still have no knowledge of the disease which produces the insanity.

For the purposes of clinical study, mental derangements of this class are conveniently divided into sub-groups, which in default of better names may be known as Complete and Incomplete Insanities.

Complete Insanities are those in which the disorder is wide-spread, involving all the faculties of the mind. The delusions in these cases are usually unsystematized.

Incomplete Insanities are those in which the whole mental mechanism is not involved, although the mental anomalies may

be so pronounced as to dominate the thinking as well as the life of the individual. The delusions are usually systematized.*

COMPLETE INSANITIES.

Insanities of this group may be divided for clinical study into those in which the attacks are single in the individual, or, if recurrent, have no definite time-relations with one another, and those in which the periods of mental aberration recur repeatedly at more or less regular intervals. The terms Non-Periodic Insanities and Periodic Insanities may be employed to designate these groups of cases.

The distinction between these groups is important, because non-periodic insanities are not necessarily the outcome of an original vice of constitution, and are often recovered from; whilst periodic insanities are the expression of an original imperfect organization or development in the brain of the patient, who rarely, if ever, gets completely well.

COMPLETE NON-PERIODIC INSANITY.

Insanities of this group may, in order to facilitate discussion, be usefully divided into three groups, on the basis of the emotional conditions. The division is given below.

It must be remembered that this classification is not offered as separating diverse diseases, but as affording an easy means of recognizing clinical symptomatic groups representing affections of whose pathology we have no distinct knowledge.

* In separating these two groups I desire to re-aver my belief that the classification is practical rather than scientific, and that there are gradations between the most severe complete insanities and the most partial form of the disorder, so that we may, at times, be at a loss to know in which class an individual case of insanity belongs. There is, however, usually no difficulty in arranging the cases. A very important difference which has been dwelt upon by recent alienists as distinguishing cases of the two classes is in the alleged fact that in complete insanities the delusions are always unsystematized, and in the incomplete they are systematized. I have already stated (see page 432) that the distinction between systematized and unsystematized delusions is, in my opinion, to some extent an arbitrary one, and that in nature every gradation between the thoroughly systematized and the unsystematized delusion may be met with.

Emotional State.	*Form of Insanity.*			
Exaltation.	Mania	Acute.		
		Chronic.		
Depresssion.	Melancholia	Melancholia.		
		Katatonia.		
Apathy from loss of activity, normal, or variable.	Mental Deteriorations.	Imbecility.		
		Primary Dementia.	Organic.	Senile.
				Hebephrenia.
			Developmental.	
			Miscellaneous.	Shock.
				Primary Confusional Insanity.
				Stuporous Insanity.
		Terminal Dementia.		

Mania.

Mania is a mental condition in which there is an emotional exaltation, accompanied by illusions, hallucinations, delusions, great mental and physical excitement, and a complete loss of the inhibitory power of the will: in acute cases, and frequently in the chronic forms of the disease, there is a marked destructiveness and a tendency to violence.

Acute Mania.—Acute mania is sometimes developed with great suddenness; more often it is preceded by a prodromic stage of emotional depression. The depressive stage may last from a few days to three months. There is often a sense of lassitude, with inability or disinclination to work, a lack of the usual power of fixing the attention, depression of spirits, and a feeling of unrest which causes the subject to worry perpetually about himself; the bowels are costive, the appetite is poor, and dyspeptic symptoms are often troublesome. The resemblance of this condition to a mild melancholy is so close that it is frequently spoken of as the melancholic stage of mania. Usually, after a time, the subject gradually returns to his normal state, except perhaps that his perceptions are abnormally quick, and that he is abnormally happy or even gay. Rapidly now the emotional excitement rises, delusions and hallucinations appear, and the maniacal stage is reached. When the affection is at its height its victim raves incessantly, shouting out a perpetual stream of incoherent threatenings, revilings, obscenities, and blasphemies. With a prodigious and untiring strength he rushes about his apartment, struggles with his attendants or his mechanical restraints, tears into shreds whatever clothing he can lay his hands upon, destroys

all about him that is breakable, smears his excrement over his person and surroundings, and so passes whole days and nights in unceasing fury. Even if for brief intervals sleep comes, it is filled with dreams, and is broken and fitful. The hallucinations and unsystematized delusions are constantly changing. There is usually great sexual excitement, as shown by satyriasis or nymphomania. There is often a marked blunting of sensation, so that the maniac does not feel the wounds he inflicts upon himself in his blind fury.

In very many cases of acute mania the symptoms are milder, but of similar character to those just detailed. Restless, licentious, blasphemous, incoherent, obscene, the maniac looks the fury of the previous picture; or, occupied by his own hallucinations and delusions, he may be rapt in a delirium of enjoyment. In a still milder form, acute mania shows itself in incoherence, irrationality, restlessness, evidences of hallucinations and delusions with marked insomnia, and total loss of modesty and of care for or notice of the usual relations of life.

In the mildest possible form of the disease—*Hypomania*—the hallucinations may be wanting, and the mania reveal itself only in a change of character, a peculiar egotistic hilarity, perpetual extravagances, restlessness, increased sexual appetite with lessened control of the will-power, leading to great sexual excesses, and a tendency to brutal violence. The diagnosis in these cases is often very difficult, and can be made only by noticing the complete alteration in the life, disposition, and mental, moral, and physical habits of the individual. Indeed, I believe that the maniacal state grades insensibly by rare cases into the normal condition, and that there are states in which the will still exerts its control, but the mental and moral attributes are so altered that the man is not his natural self. Every one has his hours and sometimes days of exaltation, and exactly when or how far the mood triumphs over the individual who shall say?

In most cases of acute mania periods of excitement alternate with periods of comparative calm. The usual duration of the disease is from three to six months, although recovery may occur in a few days or be postponed for over a year. Death may take place from exhaustion; or the mental aberration may pass into chronic mania, or into a condition of slight mental

impairment. Complete recovery occurs in about seventy per cent. of the cases.

Under the name of *transitory frenzy* is described a symptomatic affection which in the early hours of the attack might easily be confounded with acute mania, and which in some cases may really be an incipient peripheral cerebritis. It is defined by Spitzka as a condition of impaired consciousness characterized by either an intense maniacal fury or a confused hallucinatory delirium, whose duration does not exceed the period of a day or two. It will be seen at once from this definition that the only distinction which separates this alleged disease from acute mania on the one hand and acute cerebritis on the other is the rapid recovery. The symptoms also do not differ from those of an epileptic frenzy. The attack, however, differs from ordinary epileptic mania in occurring only once in the life of the individual. I do not think that transitory frenzy should be considered as a distinct affection, but only as an attack of acute violent mania, produced by unknown and probably varying causes. It would be, to my mind, just as rational to erect into distinct diseases the occasional epileptiform convulsions which occur without our being able to discover their cause, as to consider transitory frenzy a distinct affection.

Chronic Mania may develop as the result of an acute mania, or may come gradually without a preceding stage of violence. It is a condition of general mental aberration characterized by the presence of varying or non-systematized delusions, and by a condition of exalted emotional excitement. In most cases the chronic maniac, although more or less disturbed intellectually all the time, suffers from irregular exacerbations, in which the condition of excitement may become extreme and the symptoms rise in severity until they resemble those of an original acute mania. During these paroxysms, and often indeed in the intermissions, there are incoherence of speech, lack of power of association of ideas, delusions, often increased activity of the perceptive faculties with hallucinations, and mental and physical excitement. The symptoms of chronic mania are similar to those of acute mania, but are less severe in type. They are also modified by the progressive failure in the intellectual power as the patients

drift towards dementia. The hallucinations and delusions are unfixed, constantly changing, are not systematized, usually are conformed to the emotional excitement of the patient, or, if they should take for the moment a depressive or disagreeable form, do not affect the mood of the individual. They may be concerning any conceivable person, thing, or place, or may take forms not reached by the most vivid imagination in its sane moments. The moral sense is completely altered or abolished: those to whom the individual had previously been attached become objects of hate; modesty there is none, the patient revelling in obscene speech and immodest gestures, and often suffering from sexual fury. Like acute mania, chronic mania varies greatly in its intensity: indeed, the mild form of mania known as hypomania is especially apt to pursue a slow course. Chronic mania not rarely changes into chronic melancholia; whether primary or secondary, it is of long duration. Occasionally recovered from, it usually terminates in from two to five years in dementia.

Melancholia.

The connection between the depressive emotions and the health of the abdominal organs is too well recognized to need comment. There is a condition in which oxalate of calcium is found in the urine, associated with a great deal of emotional depression, some mental sluggishness, and a certain amount of bodily weakness, and occasionally, although not always, dyspeptic symptoms. This condition, which is known as *oxaluria*, is largely relieved and frequently cured by the free administration of nitro-muriatic acid, with an occasional purgative, especially if these remedies be aided by free exercise and abstinence from the use of sugar. These cases of *oxaluria* might be considered a mild form of melancholia, but, according to the studies of Dr. G. D. Stahley (*Medical News*, June 5, 1886), oxaluria occurs proportionally as frequently in other cases of insanity as it does in melancholia, and in melancholia it may at times be present and at times absent without change in the mental condition. It would appear, therefore, that there is no direct relationship between severe cases of melancholia and oxaluria.

In fully-formed melancholia the basal condition is a profound emotional depression. In a proportion of cases of so-called mel-

ancholia there is not a complete intellectual insanity, but merely an emotional depression. The patient fails to be interested in the life around him, not because he is incapable of understanding the problems of life, but because nothing but himself is of interest to him or occupies his thoughts. In the lighter degrees of the affection the patient will simply say that he is horribly depressed and cares for nothing. He sits all day in a chair, quiet, perhaps with the hands folded, seemingly thinking of nothing, with an expression of perfect indifference and apathy on his countenance. There is no interest in business, because the interest is all the time centred in himself. There is no interest in wife and family, not because the relations are not recognized, but because the man is absorbed in nursing the phantom spirit which oppresses him.

When the symptoms are more active and severe, instead of simple apathy, there is wringing of the hands and perpetual moaning and lamentation, not for any definite reason that the patient can assign, but simply because of the depression of spirits. Under these circumstances it will be found that all his thoughts are tinctured with this emotional depression. If the man is a business-man, he sees nothing but ruin before him. If he has a conscience which is not void of offence, the memory of his past misdeeds, like a Nemesis, forever haunts him. If his children are ill, they are going to be swept away by death. The whole landscape is covered with a black cloud, which throws everything into the darkest shadow. Nevertheless, there may be even yet no intellectual delusions. When the patient is aroused he talks well and reasons well. If you can get him to forget himself for a moment, his intellectual actions are perfect. After a time delusions make their appearance. They are in typical cases always unsystematized. They usually develop gradually, and not rarely are the outcome of some real feeling which the patient has. They may exist with or without hallucinations. Both hallucinations and delusions always take the depressive type. Hallucinations of hearing are the most frequent. The patient hears voices, but they are evil voices. Those who have committed murder have sometimes asserted that they had two voices in them, one crying, "Kill! kill!" the other voice trying to restrain them. Men have held their hands in the fire until they were burnt black,

because they have heard voices telling them that it was better to enter into the next world maimed than to go with a whole hand guilty of blood or other offence.

Sometimes hallucinations of sight occur; but these are less common than hallucinations of hearing. Troops of spirits from the other world pass before the patient, but it is never angels or spirits from heaven, but always sights of sorrow and of woe.

Delusions of touch are rare, and delusions of smell are still more uncommon. I do not recall a case in which I have seen a patient with delusions of smell. They, however, are occasionally present. The melancholic never smell pleasant odors. It is always sulphurous vapors or horridly fetid exhalations that oppress them.

There are certain varieties of melancholia which need brief mention. When there are no delusions the cases are sometimes spoken of as *Simple Melancholia*, as *Melancholia without Delirium*, or as *Hypo-melancholia*. In this form of melancholia, although the mind of the patient may be clear, suicidal and homicidal impulses are very frequent.

Melancholia Agitata is that variety in which there is great excitement, the patient being continually on the move, rushing up and down, lamenting loudly, wringing the hands, tearing the hair, destroying his clothes, etc. The agitation may rise to the point of complete frenzy. The melancholic frenzy differs from that of mania in being founded upon a state of intense terror and fear. The patient assaults by-standers as a matter of self-defence against their supposed machinations or attacks. Homicide, suicide, and self-mutilation are very frequent during the outbreak of melancholic frenzy.

Melancholia Attonita, or *Melancholy with Stupor*, is the variety in which all the physical as well as the mental powers of the patient are overwhelmed by the emotional depression: he is, as it were, paralyzed and dumb under the power of his fear. Lying in bed with the eyes open, or more rarely closed, asking for no food, giving no heed to any personal desires, but living in a continuous state of absolute wantlessness, he exists as an automaton. If taken up and dressed, he remains sitting in the same indifferent hopeless passivity.

The physical condition in acute melancholia is always that of

depression; the bowels are usually constipated, the breath is foul, the tongue heavily coated, the surface of the body cool, the pulse feeble, slow, or sometimes rapid, and the skin harsh. In the acute cases there is often rapid emaciation.

In certain forms of cerebral syphilis with stupor the symptoms may for a time resemble those of melancholia attonita; but the diagnosis can usually be made out by the history of continuous headache, or the presence of some evidence of a focal lesion.

Melancholia may end in death, recovery, or terminal dementia, or may become essentially chronic. It is very unusual for it to last less than three months, and I have known it to last unchanged for many years, and then the patient rapidly recover. From fifty to sixty per cent. of the cases recover. Of all the varieties of melancholy, melancholia attonita is most apt to end in dementia.

Katatonia.—A few years since, Dr. Kahlbaum, of Görlitz, separated from melancholia, under the name of *Katatonia*, a class of cases which are now believed by many alienists to be distinct. The disease is defined by Spitzka as follows:

"Katatonia is a form of insanity characterized by a pathetical emotional state and verbigeration, combined with a condition of motor tension.

"The illness begins with an initial stage, resembling that of an ordinary melancholia. This is followed by a period in which the patient presents an almost cyclical alternation of atony, excitement of a peculiar type, confusion and depression, which finally merges into a state of mental weakness approaching, if not reaching, the degree of a terminal dementia. Any single one of these enumerated phases may be absent.

"The excited stage presents symptoms of a kind different from those of ordinary melancholia, and constitutes a connecting link, as it were, between the symptoms of an agitated melancholiac and those of a lunatic with fixed delusions. Some of the patients present exaggerated, others diminished, self-esteem, and not rarely does the developing delirium assume an expansive tinge. But all katatonics exhibit a peculiar pathos, either in the direction of declamatory gestures and theatrical behavior, or of an ecstatic religious exaltation. Frequently the patients wander about, imitating great actors or preachers, and often express a desire and take steps to become such preachers and actors."

The hallucinations of katatonia are always depressive and accompanied by a melancholic depression of spirits, which is said, however, never to be so painful as in melancholia. Severe occipital headache and cataleptoid attacks are asserted to be characteristic. The cataleptoid condition is typical and extreme, the patient remaining for long periods corpse-like and immobile. I have seen two cases which perhaps ought to be classed as katatonia. Whilst under observation there was no headache and no period of excitement; but the cataleptoid condition was very marked. For hours the patient would remain standing or sitting, perfectly immobile in whatever position he might be placed. Certain forms of melancholia attonita resemble katatonic insanity, and I do not see how the diagnosis could be made between melancholia attonita and a katatonia from which the stage of excitement was wanting. Masturbation is alleged to be very frequent in katatonia, and was markedly present in one of my cases. It is, however, very common in all classes of mental weakness approaching dementia.

Mental Deteriorations.

In its fullest development dementia is that condition in which all the higher cerebral functions are abolished, so that neither thought nor emotion remains, and the individual, reduced to an automaton, simply eats when fed, and breathes when air is presented to him. The animal functions go on uncontrolled by the will, the bladder and rectum are evacuated when full, or if, as is often the case, the general nervous power is reduced to a minimum, the urine and fæces dribble over when their natural receptacles are full,—the perpetual dropping of urine indicating a distended bladder, and a constant passing of small quantities of fæces a distended rectum.

The approach to dementia is usually gradual, and death often occurs before the lowest degradation is reached, so that in the majority of cases more or less mental activity exists. The emotions are apt to survive the intellectual faculties, and, uncheeked by the will, whose power is gone, may even be unduly active. Irritable, brutal, more unreasoning and hence more unreasonable than a brute, the dement may be a most dangerous lunatic. Not

rarely, however, gentle, easily led, because without capability of intellectual persistence, he lives a childish existence.

In absolute dementia there are no grades, but the gradual courses of nature and the necessities of discussion have caused the term to be widened in its use, until now it is employed to signify simply any mental defect which is so serious as to paralyze the thinking ability of the individual.

A dementia which has been congenital or has developed in infancy is usually spoken of as *imbecility* or *idiocy*. It is generally due to original lack of brain-development, and is, therefore, scarcely the result of disease, and I shall say no more about it in this book.

Dementias due to mental disease are either primary or terminal: a primary dementia is one that has developed without obvious previous mental disease; a terminal or consecutive dementia is one that follows an attack of insanity.

Primary Dementia.—Primary dementia is a condition gradually, rapidly, or suddenly developed, in which there is suspension or great impairment of the mental powers without distinct emotional disturbances. There are included under it cases which differ greatly in the cause of the dementia, as well as in the degree of the mental impairment and in the ultimate results.

For our present purposes these various cases of primary dementia are divisible into those in which the dementia is connected with obvious organic brain-disease,—*i.e.*, Organic Dementias; those in which it is connected with general developmental changes of the body, such as puberty and old age,—*i.e.*, Developmental Dementias; and those which do not belong to either of these two classes,—*i.e.*, Miscellaneous Dementias.

Organic Dementias.—In dementia of this class the memory almost invariably suffers earlier and more severely than the other mental faculties. There is, however, nothing in the dementia itself sufficiently characteristic to enable us to recognize its etiology. The diagnosis is to be made out by noting the various symptoms, other than mental, which indicate organic brain-disease. When there is severe headache, an organic dementia is usually the result of chronic meningitis, brain-tumor or abscess, or brain-syphilis; when headache is not present, the disease is ordinarily general paralysis or its syphilitic counterfeit.

Developmental Dementias.—In this class I propose to consider two mental deteriorations,—Senile Dementia, and Hebephrenia, which respectively are associated with old age and puberty.

In some cases of old age the mental faculties are preserved almost intact amidst the general physical wreck, but more frequently the intellectual powers undergo deterioration, which may even exceed in extent that of the muscular strength. To this condition the name of *Senile Dementia* is commonly given. The mental enfeeblement is sometimes accompanied by emotional disturbances which warrant our speaking of the patient as suffering from *senile melancholia* or *senile mania*, as the case may be. Delusions are very frequently present, even when the emotional disturbance is not marked. These delusions may take the ambitious form, but usually are depressive. According to Spitzka, they are almost always unsystematized, but I have certainly seen them very thoroughly systematized. Frequently some master-passion of the individual seems in old age to increase rather than lose in force, until at last it dominates the whole character, a result which is greatly facilitated by the gradual weakening of the will. Not rarely a moral change occurs: he who has been during life chaste and refined becomes coarse and filthy in language as in person. A peculiar sexual excitement is on occasions present, giving rise to "senile satyriasis," which may lead to indecent assaults, but more frequently is manifested in the contraction of absurd or incongruous marriages. Delusions of persecution are very frequent, and the subject may live in perpetual fear. Senile dementia develops gradually, and a recognition of its earlier stages is frequently a matter of the greatest importance when there is no emotional disturbance. The first symptoms of important change are usually a loss of memory for recent events, with a loss of power of perceiving the relative importance of things and affairs. So long as the memory is fairly preserved and sufficient intellectual balance exists for the proper weighing of events, the medical expert should be very slow in deciding that the aged person is legally incompetent unless distinct delusions exist. It must, however, be borne in mind that the weakening of the will and the perversion or increase of some of the emotions render the aged especially liable to be improperly controlled by designing persons.

Hebephrenia, or *Insanity of Pubescence*, is defined by Spitzka as characterized "by mental enfeeblement marked by a silly disposition, following a preliminary period of depression, which has the same tinge as, without the depth of, that characterizing that of melancholia, and which coincides with or follows the period of puberty."

This form of intellectual aberration might very well be classed as a variety of melancholia, but I have preferred to put it in this place because in the cases that I have seen the emotional state has been one of indifference and apathy rather than of acute depression.

The psychosis may develop gradually or slowly. There is a condition of restlessness and disinclination to mental labor, combined with a line of conduct best described as silly. There is usually a distinct alteration of character. Rapid emotional changes resembling those which are so frequent in hysterical subjects sometimes take place. Paying no attention to business, abandoning lucrative pursuits, or wandering from position to position, with a constantly-increasing egotism and even a peculiar sentimentality, the subject of hebephrenia slowly loses mental power. Even at this stage sudden furious maniacal outbreaks occur, and when the loss of mental power becomes extreme these outbreaks are more frequent. Hebephrenia in the majority of cases, if not in all, rests upon a foundation of originally faulty nervous organization, and is, therefore, a protracted psychosis, which is rarely, if ever, completely recovered from. It may pass rapidly into a terminal dementia, or the patient may long remain in a condition of marked mental enfeeblement. A few cases occur in which permanent improvement has followed treatment. In a large proportion of cases hebephrenia is connected with excessive masturbation: hence by some writers the insanity is spoken of as the *Insanity of Masturbation*.

Miscellaneous Dementia.—In this division are included three classes of cases,—Dementia of Shock, Confusional Insanity, and Stuporous Insanity.

Dementia of Cerebral Shock.—There are various recorded cases in which a sudden emotional excitement has produced complete loss of the intellectual faculties: as in a case recorded by Bucknill and Tuke, in which a young lady of refinement and edu-

cation was assaulted and raped by a band of ruffians and became at once a speechless idiot for life. In a second case a young lady having by mistake fatally poisoned her father, from the time of his death "was lost to all knowledge or notice of persons and occurrences around: food she never took except when it was placed upon her tongue: the only sound which escaped her lips was a faint yes or no."

Primary Confusional Insanity is a form of mental aberration in which there is confusion of ideas and marked incoherence of speech without decided emotional disturbance. This condition may develop acutely as the result of an emotional shock, or of a cerebral overstrain, or of an exhausting disease, and may be looked upon as a milder form of dementia of cerebral shock. It seems to be a simple condition of intellectual exhaustion. It may develop immediately after the strain, or there may be a few days of incubation. Hallucinations and delusions unstable and even contradictory in character are frequent. The memory may be affected so that the patient does not recognize old acquaintances or familiar places. Delusions of identity are stated by Spitzka to be very frequent. According to the same authority, the speech affords the most characteristic symptom, the sentences being left incomplete because the subject is unable to follow an idea to its completion. Thus, a patient said to Dr. Spitzka, "I am I—I don't know that—I—is dead—funerals are—how do you do—met you in Boston steamer—this is London—London—I am sure of it—see! I have not forgotten everything—there are not so many now."

Stuporous Insanity, Acute or Primary Dementia of many English writers, *Primary Curable Dementia* of some German writers, is an affection which usually comes on in young adults, and which so closely simulates melancholia attonita in its symptoms that it is scarcely to be distinguished from it. There is, however, no distinct emotional disturbance; but if the apathy be extreme it may be impossible to determine the existence or non-existence of depressive emotions, and I believe that the two mental states grade into each other. The symptoms may come on gradually or suddenly. At the height of the attack the patient is immobile, insensitive, absolutely apathetic, sitting or lying as placed, with no wants and apparently no perception of surrounding objects.

In some cases this condition becomes so extreme that even the reflexes are affected, and in feeding the patient it is necessary to put the food well back into the pharynx. The same lack of energy is shown in all the involuntary muscles: the heart's action is slow and feeble; the bowels are obstinately constipated; the extremities are cold, and the feet œdematous, as the result of vaso-motor weakness. The urine is rich in phosphates, and the physiological discharges of the skin and uterus are suppressed. I have seen symptoms exactly resembling those of stuporous insanity as laid down in the books produced by gouty atheroma of the cerebral vessels (see page 458), and similar cases have been described by Voisin. In dementia depending upon disease of the cerebral vessels recovery rarely, if ever, occurs. Stuporous insanity has been produced by emotional shock, excessive overwork, and various causes of exhaustion, such as starvation, profuse hemorrhage, or exhausting discharges. It is also not rarely closely connected with excessive masturbation. It seems to me, therefore, that we can scarcely consider it to be other than an intense degree of the so-called primary confusional insanity. The prognosis is stated to be highly favorable, ninety per cent. of the patients recovering in a period of time which varies from three weeks to three or four months. The prognosis is less favorable when there is excessive masturbation.

Terminal Dementia.—Almost any form of active insanity may be followed by a stage in which the mind is so far lost that even the distinctive characteristics of the original insanity have more or less completely disappeared. This state is the so-called secondary or terminal dementia. The completeness of the mental ruin varies: in some cases, apathetic, mindless, without thought or emotion, the individual lives on, a mute, almost motionless, vegetating automaton; in other instances, restless, full of obtrusive or destructive activity, noisy, with incoherent talk, the dement, although overflowing with animal spirits, and perhaps, also, possessed by a peculiar aggressive egotism, is useless for any purpose,—mayhap is almost uncontrollable and exceedingly troublesome. Sometimes the mental condition is simply that of weak-mindedness, and the harmless imbecile seems like an overgrown child. Not rarely a little intellectual power remains; and if with this there be docility, the dement may be usefully employed about a farm, in the wards

of a hospital, or in other situations in which he can be carefully watched over and constantly directed and taken care of.

PERIODIC INSANITIES.

Periodic insanities are naturally divided into those in which the attacks take the form of mania; those in which they are melancholic; and those in which mania and melancholia alternate in regular cycles. These forms are respectively known as Periodic Mania, Periodic Melancholia, and Circular Insanity.

The attacks of *Periodic Mania* often begin abruptly, but may be preceded by prodromes, such as emotional depression, vertigo, neuralgia, etc. During the active stage there are hallucinations, delusions, violent excitement, furious outbursts of anger, and a pronounced tendency to impulsive actions, such as causeless assaults, indecent exposure of person, attempts to rape, etc.: in a word, the symptoms of the active stage do not differ from those of ordinary acute mania.

The attacks of *Periodic Melancholia* are similar to those of ordinary melancholia, with a pronounced tendency to impulsive acts, especially to suicide.

In *Circular Insanity*, or *Cyclothymia*, the cycles vary in length from a few days to many months: as a general rule, the more violent the symptoms the shorter is the time required to complete a cycle. The arrangement of the cycle varies in different individuals, but is constant in the same case. In this way a melancholia may be followed by a mania, and this by a lucid interval, or the mania may first appear, or the lucid interval may follow the melancholia. The passage from one mental condition to another may be abrupt, but more commonly it is gradual. The mania may be violent, resembling in all its symptoms an attack of ordinary acute mania. It may be mild, or it may even simply amount to a condition of mental exaltation, in which the subject is dominated by all sorts of immoral impulses and tendencies, which lead to a line of conduct that has been aptly spoken of as insanity of action. In like manner the melancholia varies in intensity from the most profound, hopeless, despairing apathy to a slight depression of spirits. Sometimes the lucid interval is wanting, and mania follows melancholia and melancholia follows mania in perpetually-recurring alternation. These cases constitute

the *folie circulaire* of Falret. There are certain cases in which the symptoms of a circular insanity are so slight that the patient does not at any time, to the eye of the ordinary observer, overstep the bounds of sanity. Such individuals are avoided by their friends as moody and unreasonable: to-day sanguine, talkative, energetic, and extravagant, to-morrow they are taciturn, apathetic, or full of vain regrets for acts that they have done or enterprises that they have entered upon while in the condition of exaltation.

In most cases of periodic insanity the patient during the lucid interval will reveal to the experienced observer evidences of abnormal mental action. Sexual perversion, morbid fears and morbid impulses, excessive excitability, moral degradation, sexual excesses, loss of self-control, inordinate development of avarice, jealousy, or other passion,—these are among the most common manifestations of cerebral aberration during the lucid periods of periodic insanity. According to Spitzka, kleptomania is commonly a symptom of the lucid intervals of a periodic insanity.

Periodic insanity rests upon an original faulty organization of the nervous system, and is generally hopeless. In these respects it is closely related to the partial insanities. When the lucid intervals are long and with marked mental irregularities, the disorder might well be classified as a partial insanity with regular exacerbations.

INCOMPLETE INSANITY.

Partial Insanity, Reasoning Mania, Mania without Delirium, Monomania, Mania of Character.—These terms have been employed by various writers to designate a large class of chronic insanities in which the insane condition is limited, at least in its marked manifestations, to certain portions of the brain-functions. The best scientific definition of monomania that I know of is that given by Spitzka. He says, "Monomania is a chronic form of insanity, based on an acquired or transmitted neuro-degenerative taint, and manifesting itself in anomalies of the conceptional sphere,—*i.e.*, the sphere of thought,—which, while it does not destroy entirely the mental mechanism, dominates it." This definition is, I think, scarcely wide enough. I believe that the term monomania should include not only those cases in which the intellectual or conceptional sphere is involved, but also those in which the emotional condition is affected. Avarice, envy, vanity, and

other passions are as much cerebral functions as is the reasoning power itself. There are cases in which these emotions become so magnified in their power that they dominate the whole individual. As an example may be taken the miser, in whom avarice has grown until it has entirely subjugated the ego, so that the man perishes of hunger, gloating upon the useless gold which he clutches in his hand.

The cases of partial insanity naturally group themselves into two classes: in the first of these are comprised those cases in which the delusions are distinct and apparent; in the second are included cases in which the delusions are altogether wanting or are very obscure, the insanity being confined chiefly or altogether to the emotional and moral sphere. The second of these groups constitutes the *mania of character* of Pinel.

The following quotation from Morel (quoted by Hammond, *Treatise on Insanity*, p. 365) portrays very well subjects of the mania of character, or moral insanity:

"Some have great pride and ambition, and consider themselves as being destined to the performance of acts of momentous importance. No consequence, however absurd, to which their insanity leads them, shakes their confidence in themselves. Others are impelled by bad tendencies to the perpetration of the most extravagant or monstrous acts. They rebel against all family or social obligations and duties, and are constantly considering themselves the victims of misunderstanding or injustice. For the persecution of which they imagine themselves the subjects they seek to avenge themselves on their relations, their friends, and the world at large by making a parade of their immoral conduct, thinking to compromise the interests of those who ought to be dear to them by the shameful exhibition of their depravity. They go into the streets and other public places in a filthy and ragged condition. They let their hair grow, and endeavor to attract attention by all kinds of ridiculous and improper acts. Others apply their brilliant intellectual faculties, notwithstanding they are marked by an irregularity and incoherence of action, to the production of literary works of which the extent and the plan exceed the limit that it is possible for human power to reach. These works are often in their teachings contrary to public morality and feeling. They are dreamers, utopians, false guides, who

in their mental conceptions and in the results of their intelligence and imagination exhibit the same eccentricity, the same shamelessness, as in their acts."

In all cases of mania of character there is a mental inability to weigh evidence that conflicts with the dictates of the ruling passion, which almost amounts to an intellectual insanity. In the great majority of cases sooner or later delusions will appear, although careful search may be required to detect them. As an example of such a case the following history is in point. Several years ago I was called to a palatial mansion, and was met in a boudoir by a handsome young woman, perfectly lady-like and self-restrained in her manner. She said to me, "Doctor, I have sent for you under very painful circumstances, because I feel that I must confide in some one." She then went on to say that she had syphilis, that this had been given to her by her husband, and that she had had a child born in a certain watering-place, which had died of hereditary syphilis. She further told me in detail of having personally detected her husband's infidelities. I examined the woman thoroughly, but could not find the slightest evidence of specific disease. I finally wrote to the physician who had attended the woman at the birth of the child. He at once replied that the child was well formed and perfect, and that it had died of an ordinary acute infantile disease. I then assured the woman that she had not had syphilis. She expressed herself as much relieved, but was unrelenting towards her husband, who she said kept a mistress, and frequently annoyed her by communicating with said mistress at places of amusement to which he had taken herself, and by having said mistress to drive immediately behind them in the Park. She had detectives employed to watch her husband, and had a well-known lawyer engaged to superintend the detectives. At first I entirely believed the woman's story; but after some weeks I began to suspect that there was something wrong with her brain, and to watch her.

To make a long story short, my patient watched her husband more and more closely, at an expense of hundreds of dollars, and at last one night at a theatre pointed out as his paramour a lady who was well known and entirely beyond suspicion. The whole of her story was undoubtedly invented, although she herself believed it, and for a time even misled me into crediting it.

Her husband, however, believed that she was full of the devil and wanted to torment him, and never could be persuaded that she was insane. She was all the time acting against her own interest. Her husband was a man of great wealth and natural kindliness, dotingly fond of her, and would have granted her utmost wish if she had acted in a decent manner towards him.

Her intellectual powers, except in regard to her husband's infidelities, were perfect. She mingled in society, reasoned well, and did everything well, but she had this delusion. I found in this case, as is found in most cases of monomania, that clear back to childhood there had been evidences of something not quite right with the cerebral functions. The woman had always been extremely vain, wildly ambitious to shine in fashionable society, and excessively egotistical,—characteristics which frequently precede the evidences of monomania, as was well exemplified in the history of Guiteau.

Although Esquirol used the term monomania as belonging to those cases in which there is an excess of animal spirits and of the emotions, such as ambition and anger, which are related to aggressiveness and power, there can be no doubt that there are two distinct classes of monomaniacal persons,—those in whom there is a condition of emotional exaltation, and those in whom there is a condition of emotional depression, corresponding to the mania and melancholia of general insanity.

The delusions of monomania, like the delusions of general insanity, conform in type to the emotional state, and often grow out of something having a real existence. A melancholic or hypochondriacal monomaniac may have some bad feeling, which is increased in his mind until it dominates his whole life: thus, a dyspeptic symptom leads him to the belief that he has no stomach. These cases are to be distinguished from the cases of hypochondriacal melancholia by the fact that the delusion is "systematized," —*i.e.*, it is one about which the patient reasons, and which he defends; but, as has already been stated, the systematized delusion grades into the unsystematized, and hence cases of monomania grade into cases of chronic mania or chronic melancholia.

On the other hand, the line between monomaniacal insanity and sanity is an entirely arbitrary one, and cannot be fixed by any finite power. As is well known, the children of insane persons

are very apt to be more or less different from ordinary human beings. Although they perform all the duties of life, their mental or normal organization seems to be lacking in something, or to have suffered some twist. Perverse, drifting almost of necessity into criminal acts, eccentric, such unfortunates are a long series of human atoms whose faulty brain-organization separates them from their more fortunate fellows. When this separation is sufficiently wide, when the mental organization is so bad that every one can perceive that the man is the victim of his own imperfectly-developed brain, he is said to be insane. But when the unfortunate individual is a little more like the normal human being, he is looked upon simply as eccentric, perverse, or wicked, and, unloved and unpitied, drifts through life sometimes to poverty, sometimes to the hospital, sometimes to the jail, and, it may be, to the hangman's scaffold. Sanity, insanity, criminality, power over self, free will, mental attributes, these and similar terms are household words with all of us, but no man knows whence they come, or what they are, or how far the individual is master of himself or is driven by the hand of fate, as represented in the physical conformation of the nerve-cells and fibres of his brain.

As has already been insisted upon, insanity is not a disease or a distinct entity. Necessity for an arbitrary line between sanity and insanity is not of scientific but of legal origin, and when the medical expert affirms that he is unable to measure out accurately the exact degree of human responsibility he simply acknowledges that he himself is a finite being, and that the problems of life baffle his utmost thought. It has been reserved for judges upon the bench and lawyers at the bar to arrogate to themselves the attribute of infinity, whilst ministers of the gospel but too often teach that the last and highest revelation of a merciful God is that this poor, broken humanity, helpless so often in the iron grip of its own perverse nature, shall be punished by flames eternal.

The difficulty of drawing a line between sanity and insanity is well illustrated by religious monomaniacs. Are those who believe that they habitually hold communion with spirits, the dupes of modern spiritualism, to be considered insane? Is the woman who is convinced that Providence has as the result of her prayers put back the ordinary course of nature and relieved her of an incurable affection, sane or insane? In matters of religious belief,

every man who holds strongly to a certain faith might consider every person who believes in a different faith to be insane. Profound belief in the daily presence of spirits and in the utmost vagaries of modern spiritualism may coexist with great scientific or business acumen.

Although it seems impossible to fix a line which shall separate a sane from an insane religious trust or belief, it can hardly be doubted that many of the devotees of spiritualism must be considered as across the line. To illustrate how closely monomania is related to sanity, and how difficult it sometimes is to draw the line, I may cite the case of a superior officer of the United States army, now dead, who was, at the time I speak of, stationed at one of the frontier posts of the country, and was performing, with satisfaction to all, the duties appertaining to his rank. One day he said to a friend, "My life in this frontier post, from its monotony, would be absolutely insupportable if it were not for my daily mail which I get from my dead friends. I had a letter from your brother last week, and every morning I live in expectation of receiving a letter from some deceased friend or relative." He was asked how the letters came, and it was learned that they were received through a certain living medium in the eastern part of the United States. This woman, no doubt receiving a stipend from the officer, would almost daily write him a letter, which was received by him with absolute credence as coming through her from the spirit-world. On another occasion he said, "There is one thing that gives me great comfort, and that is that I am a descendant of the Virgin Mary." Some one intimating a doubt of that genealogy, he continued, "I know that I am a descendant of the Virgin Mary, because I had a letter from her, and she says so; and she certainly ought to know." Can we hesitate in deciding that here was a delusion which was thoroughly systematized and logically defended by its holder? The first premise, that he received letters from the spirits, was false, but his reasoning based upon it was sound.

Not only is it difficult to draw the line between sanity and insanity, but there is a close relation between partial insanity and high intellectual power, especially with reference to genius or the power of original thought. There can be no doubt that a proportion of those who are considered as the most pronounced

examples of genius are men whose intellects are on the border of insanity. To say that an individual is a genius usually means that he has a certain function of the mind exalted high above the other functions. A man whose imagination is developed out of proportion to his reasoning faculties is apt to be a poet or a novelist, and produces works which may live through centuries. Such a man is above his fellows, not by virtue of great brain-power, but because his brain takes a peculiar limited direction. A genius is recognized as a man not practical. By a man not practical is meant one lacking in common sense; and common sense is, after all, neither more nor less than the term used to express good judgment in the ordinary matters of life. A man who has great imagination usually lacks reasoning power, and is not practical, because of his great imagination. One of the best examples of the relation between extraordinary imaginative power and monomania is John Bunyan, the author of "The Pilgrim's Progress," which, according to any standard that we have a right to set up, is one of the five or six greatest books in the English language. It is certainly more read than any other book with the exception of the Bible. No one who reads the history of Bunyan's life can doubt for a moment that he for a long time suffered from monomania with depressing delusions, and his immortal dream may have been to him, at least at times, much more than a dream.

Space is wanting to do more than call attention to the overweening egotism of Byron and to the agonies of mental depression which overshadowed the life of Cowper; but I cannot forbear citing at greater length the case of Victor Hugo as illustrating the close relationship between insanity and genius. His uncle died insane; his brother, Charles Hugo, in his late boyhood gave promise of remarkable talent for literature, but before twenty he became insane, and finally he passed into a condition of complete dementia. One of the daughters of Victor Hugo is now and has been for many years living in an insane asylum. According to the London *Medical Times*, there are in many of Hugo's finest productions numerous passages which could have been conceived only by a diseased imagination, and which are indelibly stamped with madness. A remarkable fact in the mental history of the great French poet is that along with his extraordinary imagina-

tion there was a shrewdness almost as great. No banker could have more carefully managed his fortune; no politician could have more tenderly nursed his popularity. He who had amassed over a million of dollars died the idol of a communistic democracy; he who had played at fast and loose with all political parties was buried amidst a tumult of universal sorrow.

The prognosis in monomania is exceedingly unfavorable. This is because the condition is so often the result of a faulty formation of some part of the brain. There is a peculiarity of the cerebral organization which shows itself from the very first. Only a small proportion of cases recover entirely.

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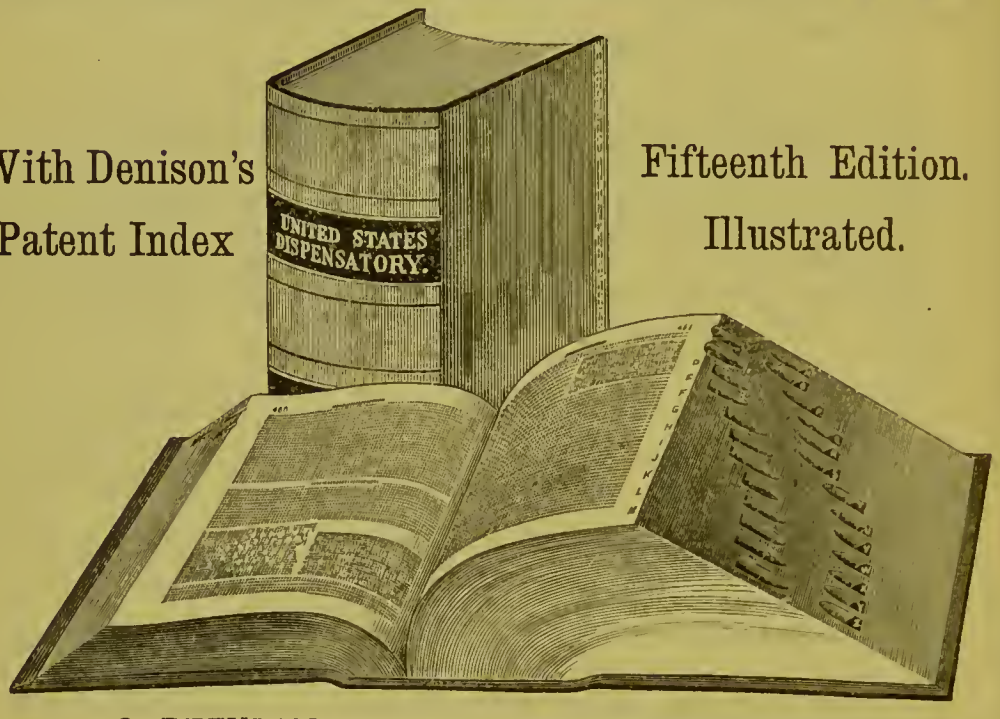
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